Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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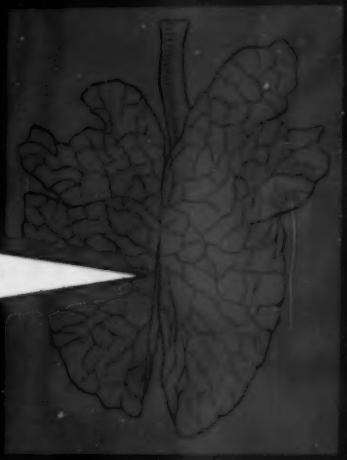

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Correspondence

Appreciation of Honorary Membership

Following are letters received from the three men elected to honorary AVMA membership at the Cleveland meeting last August.

Sept. 11, 1957

Dear Dr. Hardenbergh:

The action of the AVMA Executive Board and House of Representatives in electing me to honorary membership in the American Veterinary Medical Association is a recognition I appreciate more than I can express adequately. Knowing the pride with which your membership holds its profession, the meaning of being elected to honorary membership is all the more significant.

During the past 11 years, it has been a privilege to work closely with many key members of your profession to protect and improve the health of the livestock population of this country. It is my sincere hope that whatever service we have been able to offer in the past may be increased in the future, and that our activities will always be consistent with the objectives, ethics, and principles of the profession of veterinary medicine.

Respectfully yours,
W. D. Knox, Editor,
HOARD'S DAIRYMAN.

• • •

Sept. 13, 1957

Dear Dr. Hardenbergh:

I certainly do regard it as a great honor to have been chosen an Honorary Member of the American Veterinary Medical Association and want to express my appreciation and thanks. I also should like to ask you to forward my thanks to the Executive Board and House of Representatives of the American Veterinary Medical Association as well.

My co-workers and I regard it as a great honour and privilege that our scientific work has been estimated so highly by the American Veterinary Medical Association . . . Your recognition makes us gladly continue our work hoping that we may further depend on the kind and valuable assistance of the veterinary medical institutions and colleagues of your country . . .

With kindest regards,

Yours very sincerely,
s/Prof. Dr. E. Roots, Director,
Veterinary Hygiene and Animal Disease Institute,
Giessen University, Germany.

• • •

Oct. 9, 1957

Dear Dr. Hardenbergh:

During the recent Ninety-Fourth Annual Meeting of the American Veterinary Medical Association, your Executive Board and House of Representatives elected me to Honorary Membership in the American Veterinary Medical Association. I wish to express my deepest thanks for this honor and to state that I am proud indeed to display my certificate of membership.

I have long had an interest in veterinary medicine, stimulated from having been reared on a Midwest farm; also, because of the frequent necessities in earlier years of medical officers being called on to perform certain duties normally assigned to veterinarians; and from the fact that during the past few years my assignments in the Air Force Medical Service have required that I work closely with the veterinary service in developing the broad aspects of the military medical service for the Air Force.

Again with my sincere thanks to you and the American Veterinary Medical Association, I am

Sincerely,
Dan C. Ogle, Major General,
U.S.A.F. (M.C.) Surgeon General.

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in ketosis

the way it was

1945

"This continues to be an important problem to the veterinarian in dairy cattle practice. Some years ago, it appeared to be primarily a disturbance of such a nature that the body was unable to metabolize the fats—more carbohydrate was needed, more ready energy. Many cases responded to glucose intravenously, supported by the feeding of cane molasses or corn syrup. Others responded when chloral hydrate was administered, its effects being to liberate the glycogen stored in the liver.

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Report of the Committee on Diseases of Dairy Cattle, R. C. Klussendorf, Chairman, J.A.V.M.A. *107*:355 (Nov.) 1945.

the way it is

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Link, R. P.; Newton, D. L., and Robb, W. C.: Paper presented at 93rd Annual Meeting A.V.M.A., Oct. 15-18, 1956, San [illegible]

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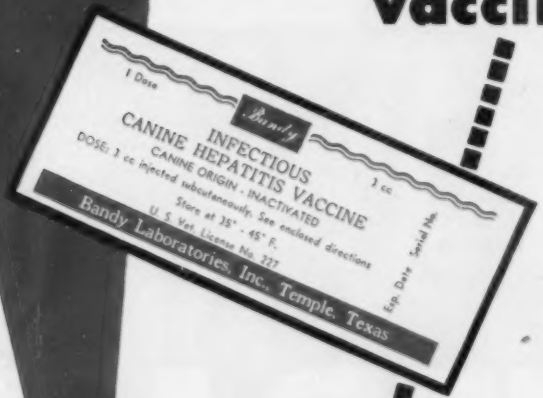

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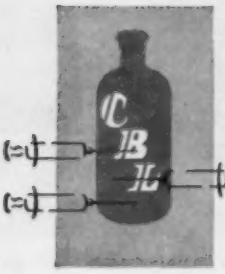

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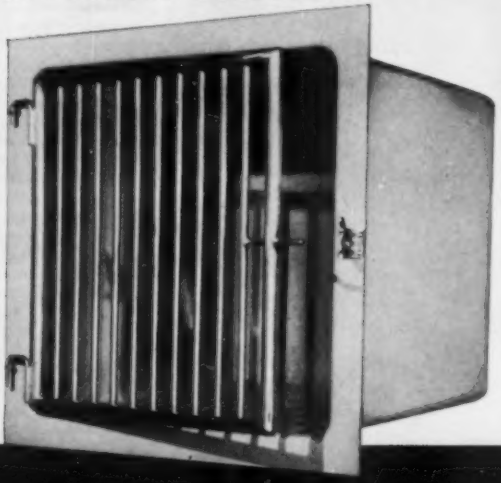

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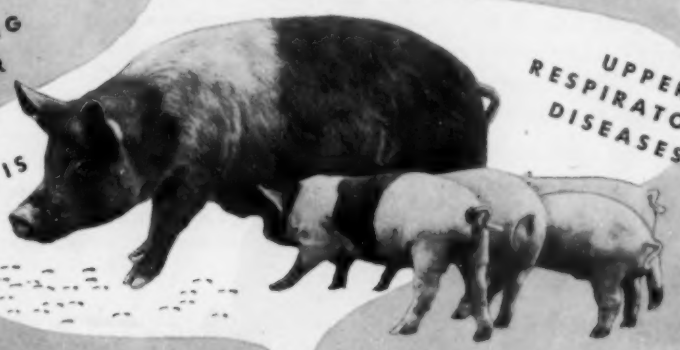

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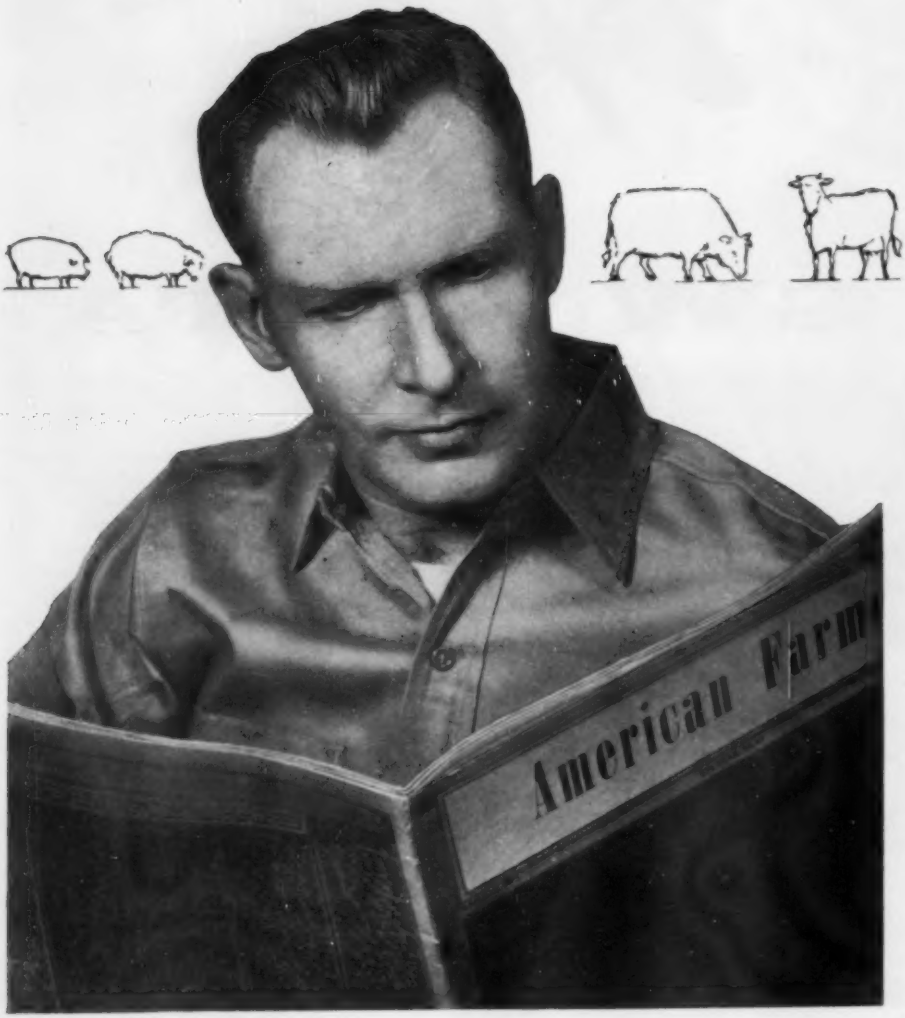

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News From Washington

The Honorable Carl Vinson, chairman of the House Armed Services Committee, has written a strong letter of protest to the new Secretary of Defense concerning the **proposed transfer of functions of the Veterinary Corps to the Department of Agriculture.**

Mr. Vinson has asked that the previous directive be cancelled and that full information be furnished to his committee.

The Honorable Richard B. Russell, chairman of Senate Armed Services Committee, protested likewise, and **requested the entire matter be re-examined,** and that a complete report be furnished that committee, including precise savings (if any), and the effect on present and future functions of the Armed Forces Veterinary Services.

★ ★ ★ ★

A number of **Army veterinary officers,** receiving orders to active duty recently, will report to the Meat and Dairy Hygiene School, Chicago, on Oct. 30, 1957, for a course of instruction (see JOURNAL, August 15, 1957, adv. p. 6, relative to authority for same). They will receive permanent station assignment upon completion of course, on or about Dec. 21, 1957.

Air Force ordered 17 veterinary officers to active duty, effective date Oct. 7, 1957. They reported to Gunter Air Force Base to take the basic orientation course.

★ ★ ★ ★

Poultry Division, AMS, held a series of meetings in selected cities during October to discuss proposed regulations relating to **the compulsory inspection program** (see JOURNAL, Oct. 15, 1957, adv. p. 6). The poultry industry, state departments of agriculture, public health, consumer groups, and other interested persons were invited to attend.

★ ★ ★ ★

OASI Study Council Named.—A 12-man advisory council will review the financial position of the Social Security System and report back to Secretary Folsom. The council, provided for by the Social Security amendments of 1956, will review the status of the OASI trust fund, and the new disability insurance trust fund in relation to the programs' long-term obligations. Council members are: (representing employers) Elliott V. Bell, editor of **Business Week;** Reinhard A. Hohaus, Metropolitan Life Insurance Co.; Robert A. Nornby, Pacific Lighting Corporation; (employee representatives) Joseph William Childs, United Rubber, Cork, Linoleum & Plastics Workers; Nelson H. Cruikshank, AFL-CIO; Eric Peterson, International Association of Machinists; (representing the public and self-employed) J. Douglas Brown of Princeton; Arthur F. Burns, former head of President Eisenhower's council of economic advisors; Carl H. Fisher, University of Michigan; Thomas N. Hurd, Cornell University; Robert McAllister Lloyd, Teachers Insurance and Annuity Association; and Malcolm H. Bryan, Atlanta Federal Reserve Bank.

★ ★ ★ ★

For the fourth year, a postgraduate course on "Pathology of Diseases of Laboratory Animals" will be given at the Armed Forces Institute of Pathology, Washington, Dec. 9-13, 1957.

The course will provide training for professional officers who are responsible for the recognition and interpretation of lesions of experimental animals, or who have charge of procurement and maintenance of animal colonies. Application for enrollment from civilian physicians, dentists, and veterinarians should be sent to the Director, Armed Forces Institute of Pathology, Washington 25, D.C.

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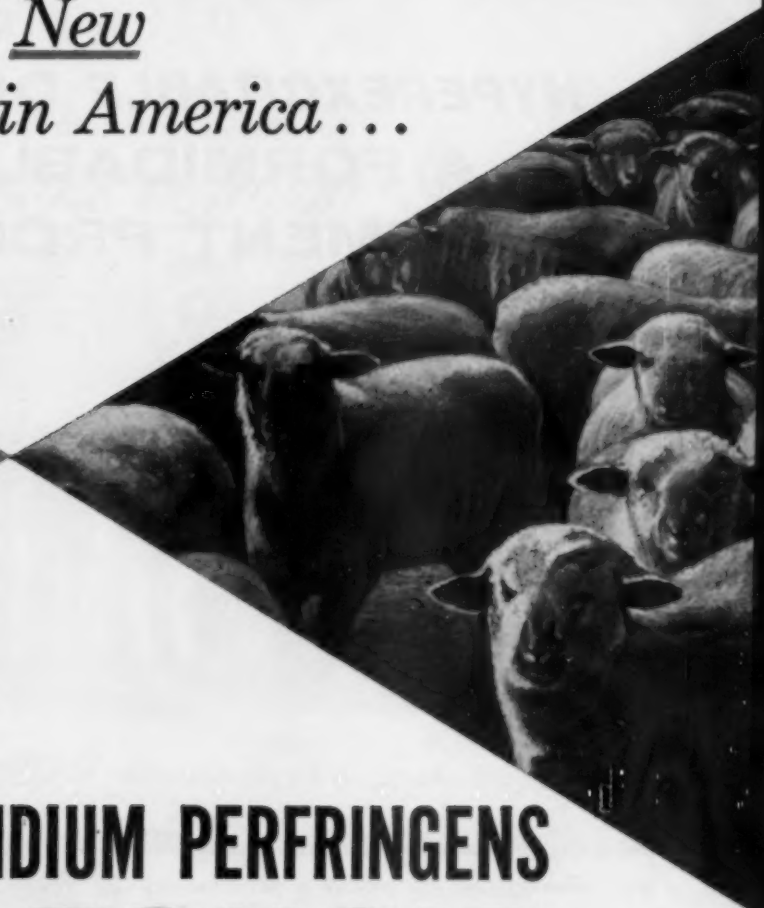

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REFERENCES: 1. Bull, W. S.: N. Amer. Vet., In press. 2. Henry, R. T., and Blackburn, E. G.: Vet. Med., In press.

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Some Clinical and Histological Observations on Scrapie in Sheep

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Lafayette, Indiana, and Baltimore, Maryland

ALTHOUGH RECOGNIZED as a distinct clinical entity in Great Britain and other parts of Europe for only about a century, there is considerable evidence that scrapie in sheep existed in those countries as early as the middle of the eighteenth century.[17] Many terms have been applied to this disease by laymen of different countries, but the highly descriptive term "scrapie" has enjoyed the greatest popularity and has, therefore, been retained in English-speaking nations. The term is derived from the Scottish word which refers to the rubbing exhibited by the affected sheep. Characteristically, clinical features of this disease in sheep, in addition to the rubbing, are nibbling at the skin of their legs, ataxia, weakness, emaciation, paralysis, prostration, and death.

During the past 20 years, scrapie has been considered a virus infection of the central nervous system by a number of investigators. This theory was based on transmission studies in France[7] and in Great Britain.[10,19] On the basis of myopathic studies, it has been suggested that scrapie may be part of a more widely occurring myopathy, possibly hereditary in nature.[4]

The first case of scrapie on the North American continent was reported in Ontario, Can., in 1938,[16] and in 1946, 4 additional cases were reported in three flocks.[15] The first case of scrapie in the United States was reported in Michigan in 1947.[18] In 1952, scrapie occurred in two California flocks.[5] A compilation of all known cases in the United States was presented by the Animal Disease Eradication Branch, U.S.D.A., in May, 1956.[1] This report indicated that the disease had been found to exist in 17 states, involving 46 flocks in which a total of 109 cases had occurred.

Heretofore, this had been considered a chronic disease associated with degenerative lesions in certain areas of the central nervous system. A number of investigators have reported that the most significant lesions in scrapie were the presence of vacuoles in neurons, particularly in the medulla, and to a lesser extent in the thalamus, pons, and spinal cord. These neuronal vacuoles were recognized in clinical cases for many years and were described in regard to location and incidence.[2,3,6,11,17,19]

Most investigators have not reported inflammatory lesions in central nervous systems of sheep affected with scrapie. Perivascular infiltration in the brain was found in only 1 sheep, which showed convulsive signs, among 75 clinical cases of scrapie, 39 cases other than scrapie, and 59 apparently healthy sheep.[11] However, the brains of sheep with experimentally induced scrapie showed mild infiltration of lymphocytes into the pia mater in depths of cerebral sulci;[19] there was also mild perivascular in-

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TABLE I—Individual Sheep Affected with Scrapie

No.	Date received	Age (yr.) and sex	Extent of symptoms	Vacuolated neurons	No. of days observed	Disposition
1	9-13-54	3 M	Marked	+	7	Died
2	3-11-55	5 F	Marked	+	10	Killed
3	7-21-55	2½ M	Moderate	+	....	D.O.A.†
4	8-8 -55	1½ M	Marked	−	1	Killed
5	8-8 -55	2 F	Moderate	+	1	Killed
6	8-23-55	3 F	Marked	+	....	D.O.A.
7	8-23-55	3 F	Moderate	+	0	Killed
8	8-23-55	1½ M	Moderate	+	0	Killed
9	9-14-55	3 M	Marked	+	25	Died
10	9-30-55	2½ F	Slight	−	121	Killed
11	10-4 -55	5 F	Marked	+	58	Died
12	10-12-55	2½ F	Marked	+	44	Killed
13	12-8 -55	2½ F	Moderate	+	....	D.O.A.
14	3-19-56	3 M	Marked	+	23	Died
15	5-16-56	.... M	Moderate	+	....	....
16	6-28-56	2 M	Moderate	+	22	Killed
17	9-17-56	2 M	Moderate	−	36	Killed
18*	9-26-56	4 F	Marked	+	135	Killed
19*	1-12-57	2½ M	Marked	+	18	Killed
20*	1-30-57	3 F	Marked	+	57	Killed

*Animals were examined and found to have gross myopathy. Histological examination revealed degenerative changes in the affected muscles. †Dead on arrival.

filtration of lymphocytes in vessels of the brain and brain stem.

Few detailed studies of the peripheral nervous system have been reported in cases of scrapie, although the typical signs of the disease such as intense rubbing, nibbling at legs, and ataxia suggest that peripheral neuropathy may be present. Peripheral neuritis was reportedly found in scrapie[3] but this has not been substantiated. The dorsal root ganglia also appear normal.[13]

Recent investigations[4,14] showed that a type of myopathy was associated quite commonly with the classical form of clinical scrapie. The lesions consisted of degenerated muscle fibers which became enlarged and lost their cross striations. Eventually, disintegration of muscle fibers occurred. There was an increase in sarcolemmic cells.

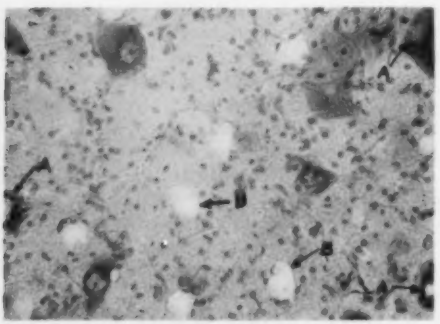

Fig. 1—Vacuoles are shown in neurons of the brain from a normal sheep; (a) vacuole; (b) capillary. x 243.

These workers expressed the opinion that vacuolation of neurons, previously described in scrapie-affected sheep, did not furnish a satisfactory explanation for the development of the clinical syndrome. Rather, they stated that the discovery of a myopathy gave this disease a satisfactory basis for the first time.

Vacuolated neurons and perivascular infiltration of blood vessels[19] in medullas of apparently healthy sheep, similar to those observed in cases of scrapie, were reported recently.[20]

It seems evident, therefore, that considerable discrepancy exists, concerning the actual site or sites of damage in this disease entity, between central nervous system, on the one hand, and skeletal musculature on the other. The divergent reports cited above raise the question of the significance of vacuolated neurons as an indication of chronic central nervous system disease. It is the purpose of this paper to describe briefly the clinical signs of the disease, scrapie, and attempt to assess from histological material the relative importance of nerve and muscle damage in the manifestations of scrapie.

Material and Methods

The first case of scrapie in Indiana sheep occurred in September, 1954. Since then, 20 animals, representing 19 flocks in different sections of the state, have shown characteristic signs of the disease. These cases furnish the material for this study. Of the 20 sheep, 14 were kept under our observation for one to 135 days, depending on their physical condition when received. Ten of the

14 were killed and 4 died while under observation. Of the remaining 6 sheep, 2 were killed at once in order to expedite histological examination of the central nervous system; 3, which had exhibited clinical signs of scrapie, were dead on arrival at the laboratory; and the head of the sixth was submitted for examination.

When a diagnosis of clinical scrapie was established in an individual animal, the entire flock was considered exposed and removed for immediate slaughter under the supervision of the state and federal officials who are responsible for eradication of scrapie in this country.

Routinely, portions of thalamus, pons, medulla, and cervical cord of affected animals were examined and fixed in 10 per cent formol, followed by paraffin embedding. Sections were stained with hematoxylin and phloxine.

The brains of 1 animal showing clinical scrapie and 2 normal adult sheep were studied more intensively. These animals were given Nembutal intravenously and exsanguinated by severing a carotid artery. After incision of the internal jugular vein, both carotid arteries were immediately perfused in a cephalad and caudad direction with 10 per cent formol. Perfusion was continued until the musculature became rigid (usually 6-8 liters). Brain, spinal cord, and attached ganglia were removed and fixed in 10 per cent formol. Celloidin-embedded serial sections were cut from 1 diseased and 1 control animal. Blocks from the second control were sectioned serially in paraffin. All sections were stained for nerve cells with gallocyanin and for neuroglia and myelin using Greenfield's[9] modification of Mallory's phosphotungstic hematoxylin.

In 3 sheep which exhibited clinical signs of scrapie, a careful search was made for macroscopic lesions in the muscles. Such areas were removed and prepared for histological study according to the methods already indicated.

RESULTS

Clinical Signs in Affected Sheep.—Ten of 14 sheep, which were kept for varying periods, had well-defined signs of scrapie which became progressively more severe during the period of observation.

Loss of wool, due to continual rubbing provoked by intense itching, occurred in

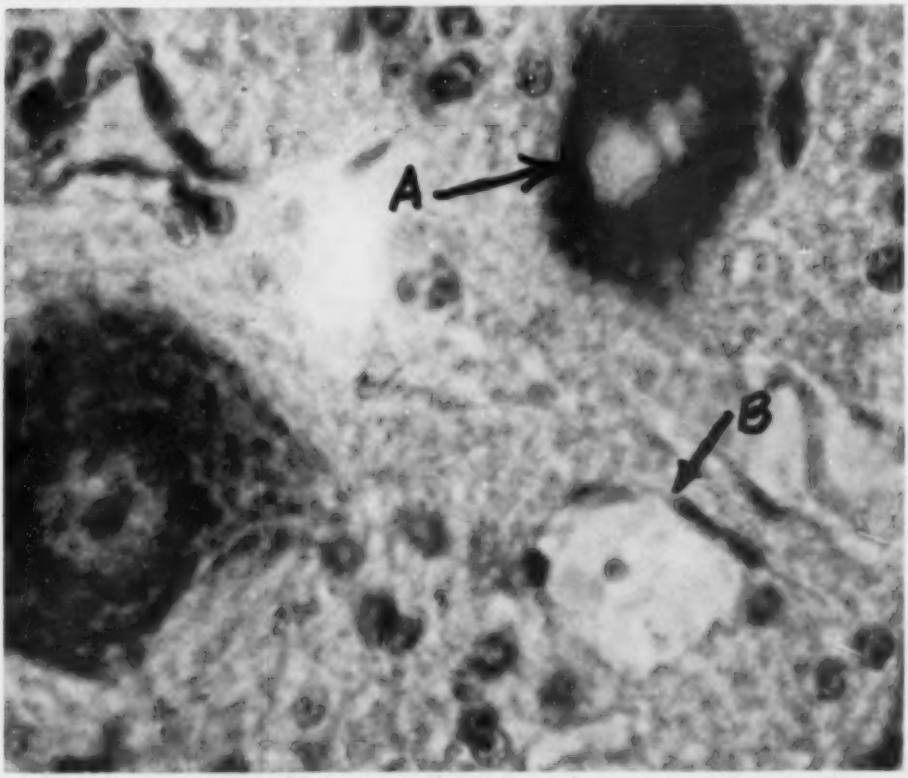

Fig. 2—This is a higher magnification of a vacuolated neuron in the same area as in figure 1. x 1,514.

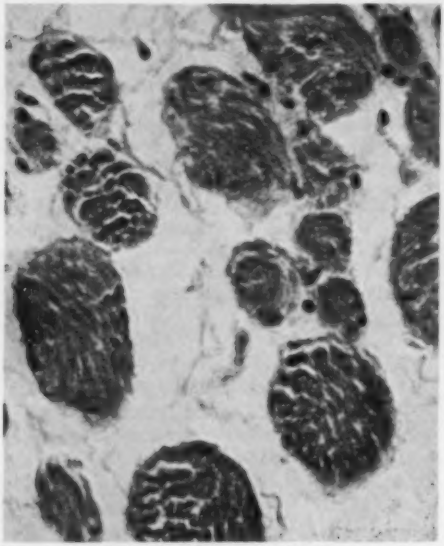

Fig. 3—Isolated atrophic muscle bundles are shown in an area where adipose tissue was replacing them. From the vastus intermedius of sheep affected with scrapie. x 677.

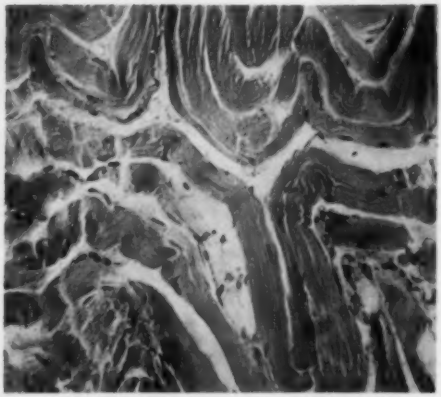

Fig. 4—Vastus intermedius muscle of a sheep affected with scrapie, showing degeneration and disruption of muscle bundles with accompanying increase of muscle cell nuclei. x 304.

patches on various parts of the body in all cases, but the depilation was more common around the ischial bones and along the flank region. Appetite, except when the sheep were nearly moribund, was not ap-

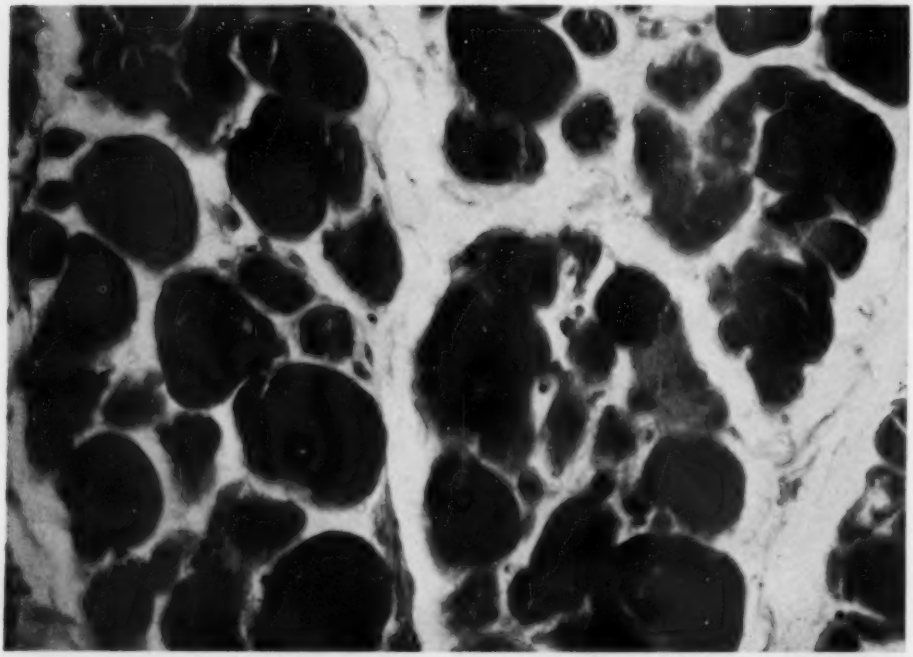

Fig. 5—Cross section of extrinsic eye muscle of sheep affected with scrapie showing degenerated and rounded muscle bundles. Note the variation in the size of the muscle bundles and the variation in affinity for acid stain. x 580.

preciably impaired. Animals in a severe state of cachexia still ate quite well. In varying degrees, these sheep also showed peculiar nibbling at the skin of the legs, rubbing of forehead and trunk regions, accompanied by quivering of the lips and rapid viper-like movements of the tongue. Motor incoordination, which is frequently mentioned in association with scrapie, was also a clinical feature observed in these sheep when they were allowed to run in an open lot.

Incidental Pathological Findings.—Pneumonic lesions were found in 1, stomach worms in 1, and nodular worms in 2 of the sheep. Postmortem changes had developed in 3 of the animals before necropsy was possible.

Histological Examination of the Central Nervous System.—Vacuoles, sometimes with peripheral nuclear displacement, were found in neurons of the medulla in 17 of the 20 sheep. They were present less frequently in the basal ganglia, thalamus, pons, and gray matter of the spinal cord. When vacuolization was extensive, multivacuolated nerve cells were frequently found. These results are summarized (table 1).

Serial sections of the brain of 1 sheep with scrapie and 2 controls were examined and compared. There was no essential difference in size, character, frequency, or distribution of single and multivacuolated neurons between the controls and the sheep with scrapie (fig. 1, 2). In these animals, representative spinal ganglia were normal.

Other than vacuolated nerve cells, no neuronal alterations were observed. The Nissl substance, including that in vacuolated neurons, was normal. There was no evidence of glial nodules, diffuse inflammatory changes, or unusual perivascular cellular infiltration. Moreover, there was no demyelinization.

Gross and Microscopic Examination of Muscles of Three Affected Sheep.—The most obvious lesions were in certain muscles of the hindlegs, particularly the pectineus, vastus intermedius, vastus lateralis, and semitendinosus. These lesions consisted of pasty moist masses, almost white in color in contrast to normal muscle tissue. Such areas were difficult to dissect away from the intermuscular fat of the muscle bundles.

Perhaps the most extensive histological lesions were found in the pasty mass removed from the vastus intermedius muscle of 1 sheep which also showed a similar involvement of the pectineus. The muscle was largely replaced by adipose tissue in which isolated atrophic bundles were present (fig. 3). In some fields, the muscle bundles showed loss of normal cross striation (fig. 4). In others, considerable connective tissue proliferation had taken place. The bundles appeared in various sizes; some were atrophic and fragmented and others hypervoluminous. In cross section, they were rounded instead of rhomboidal as in normal muscle (fig. 5). In other instances, there was nuclear accumulation in the muscle bundles. Some of these showed disruption and atrophy (fig. 6).

Fig. 6—Semitendinosis muscle of sheep affected with scrapie showing marked increase of nuclei in muscle bundles. Note weak staining of muscle fibers in center of field. x 348.

These changes were not confined to leg muscles. Although the cutaneous muscles of the trunk from 2 animals did not show myopathic changes, the musculature removed with the skin of the forehead of 1 animal revealed degenerated bundles among which there were spaces filled with fat (fig. 7, 8). In 2 animals, the muscle bundles in extrinsic eye muscles were similarly affected (fig. 9).

Gross and Microscopic Examination of the Skin.—One of the most common clinical features of this disease has been the loss

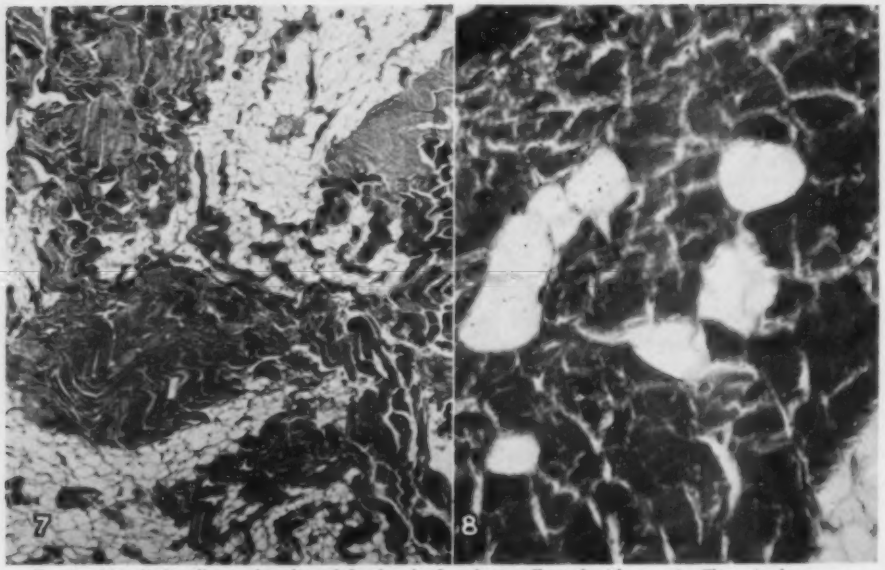

Fig. 7—Muscle bundles under skin of forehead of a sheep affected with scrapie. There is degeneration and disruption of fibers and replacement of muscle bundles by adipose tissue. Note the variation in affinity for acid stain. x 98.

Fig. 8—Same as figure 7 showing degeneration of muscle bundles on cross section and replacement of some bundles by adipose tissue. x 405.

of wool covering the skin in areas where pruritus seemed to be most intense. Aside from small papillary eruptions, the skin in these areas was apparently normal.

Microscopic examination of the skin from denuded areas of 6 affected sheep revealed aggregations of lymphoid cells around several small branches of nerves in the subcutis (fig. 10). Perivascular cuffing was also present in the same areas.

DISCUSSION

Vacuolated nerve cells in certain areas of the central nervous system, particularly in

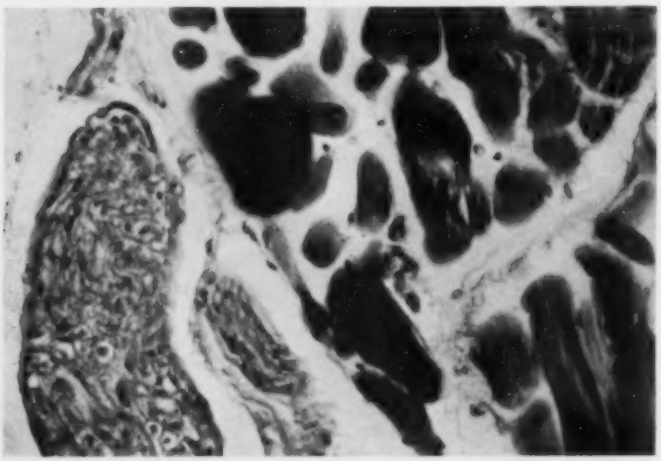

Fig. 9—Extrinsic eye muscle of sheep affected with scrapie showing degeneration and disruption of muscle bundles. Nerve bundle in lower left of field appears normal. x 405.

the medulla, have long been considered the only constant and specific pathological lesion in scrapie. Of 20 sheep with scrapie in this study, 17 showed vacuolated neurons in the medulla. In brains more completely studied, similar neuronal changes, though less frequent, were also found in the thalamus, pons, basal ganglia, brain stem, tegmentum, and gray matter of the spinal cord. However, no other neurohistological abnormalities were observed. It was reported[6] that vacuoles occurred occasionally in sheep affected with other conditions but that these were of smaller size and could only be seen in necrotic nerve cells. However, in this paper we have described vacuolization, identical in every way to that found in scrapie, in healthy sheep. It seems evident, therefore, that these histological changes can not be used as conclusive evidence of this disease.[4,20] Their presence raises certain interesting possibilities. They may occur as a normal variant in brains of healthy sheep. They may be related to terminal and agonal death factors, particularly anoxia. They may be the result of histochemical changes in nerve cells and of such things as temperature, methods of fixation, and embedding.

Vacuolated neurons not necessarily associated with specific disease have been demonstrated in other animals. It was recently reported[12] that in cats and in selected necropsy material from man the extent of vacuolization was related to the duration of critical hypoxia preceding death. Occasionally, intracytoplasmic vacuoles have been seen in nerve cells rendered ischemic by venous distention.[8]

Much more pathognomonic have been the myopathic lesions in leg muscles. The gross lesions of muscular dystrophy were similar but not as extensive as those previously reported.[4] It may be of considerable practical importance that such myopathic changes are also found in extrinsic eye muscles and cutaneous muscles. An examination of these readily accessible muscles could well be useful in the diagnosis of scrapie in sheep.

The cause of this disease remains obscure. Whether it is a purely infectious disease, a primary disorder of the neuromuscular system, a nutritional and metabolic disturbance, or whether there is a hereditary predisposition to an infection, remains unsettled. Because of the high cost of slaughtering entire exposed flocks, the cause of scrapie and a more convincing method of laboratory diagnosis must be found.

SUMMARY

Clinical signs characteristic of scrapie were observed in 20 sheep from 19 flocks in Indiana.

Histological examination of the central nervous system revealed vacuolated neu-

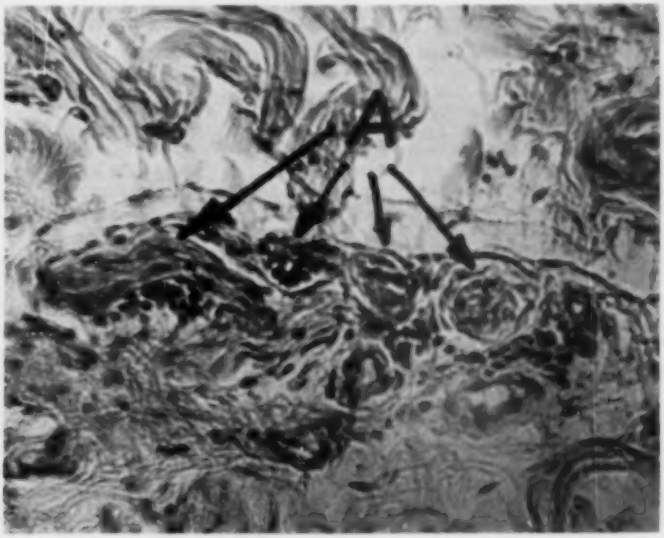

Fig. 10—Denuded area of a pelvic limb of a sheep showing lymphoid cell accumulations about small nerve bundles in the subcutis; (a) nerves. x 580.

rons, particularly in the medulla, of 17 of the 20 animals. However, equally prominent vacuolated neurons were found in the brains of normal adult sheep. No other histological abnormalties were found in the central nervous systems of either normal or affected animals.

Muscular dystrophy was found regularly in certain muscles of the hindlegs, extrinsic muscles of the eyes, and cutaneous muscles of the forehead in all 3 of the affected sheep studied. These superficial lesions could be used as an aid in the clinical diagnosis of this disease.

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Response of Sheep Nematodes to Phenothiazine—An Abstract

An 18-month study, with varying amounts of phenothiazine, was made of parasites of sheep on three central Kentucky farms.

On farm K, drenching with copper sulfate-nicotine sulfate (Cunic) was effective in reducing the fecal egg counts in every instance. Although a high average egg count developed while phenothiazine-salt was available, the drug was effective in reducing larval development.

On farm S, where drenching with phenothiazine was the sole medicinal therapy, it was effective in reducing the egg count averages.

On farm B, where the most extensive drenching program was employed, only the Cunic drenches were consistently effective. In nine instances when phenothiazine drenches were used, the average egg counts were not decreased; actually, substantial egg count increases followed in several instances. A higher percentage of the larvae developed on farm B when phenothiazine-salt was available than on farm S where salt mixture was not used. The larval development in farm B lambs, while given phenothiazine, was about the same as in farm K lambs while the drug was not given.

A phenothiazine tolerance in the worms on farm B would seem to be indicated by: (1) the extensive drenching program utilized; (2) the consistent success with Cunic drenches; (3) the comparatively high percentage of larval development when phenothiazine-salt was available; (4) the apparent ineffectiveness of the phenothiazine drenches on farm B compared with farm S; and (5) a ten-year history of

extensive phenothiazine utilization by farm B.

This observation is compatible with the controlled laboratory findings for the same strain of worms (strain B) reported earlier. While these observations are not regarded as sufficient evidence to justify general discontinuance of phenothiazine drenches or salt mixtures, it would seem to be advisable, especially in flocks with a history of parasite problems, to alternate the Cunic and phenothiazine drenches and, in addition, to keep the salt mixture available.—[*S. E. Leland, Jr., J. H. Drudge, Z. N. Wyant, and G. W. Elam: Strain Variation in the Response of Sheep Nematodes to Action of Phenothiazine. III. Field Observations. Am. J. Vet. Res., 18, (Oct., 1957): 851-860.*]

Taeniasis.—Cysticercus bovis was found at slaughter in 22 of 27 cattle from a Minnesota farm. The hired man, who had returned several months previously from military service in Europe, was the probable source. He denied having symptoms but, of 8 residents, ova of Taenia were found in his stools only.—*Pub. Health Rep., May 25, 1957.*

A Cytopathogenic Viral Agent in Feces of Cattle.—Ten days after 19 calves, 3 to 4 months old, were brought into an experimental unit, they developed a fever (104 to 106 F.), and several had a slight cough and nasal exudate. Two pregnant cows brought to the same unit developed a fever and nasal exudate within a week. One recovered in a few days but aborted one month later; the other developed pulmonary complications. In neither cow nor in the aborted fetus could evidence of infection with *Vibrio fetus, Leptospira pomona,* or *Brucella abortus* be found. Similar signs of infection, plus mucus in the stools, developed in 9 older calves later brought to this same unit.

A viral agent, cytopathic for bovine kidney cells grown *in vitro,* was recovered from the feces of several of these animals, from the stomach and placental fluids of the fetus, and from the feces of calves with a similar syndrome in another experimental unit. Feces from calves and cows with no history of illness were free of this virus.

Serums taken from 4 of the calves before infection failed to prevent cytopathic changes while serums taken during convalescence did prevent such changes. The etiological significance of this virus in the disease described has not been established. —*T. Moll and A. V. Finlayson in Science (Aug. 30, 1957): 401.*

Infectious Bovine Rhinotracheitis—An Abstract

The virus of infectious bovine rhinotracheitis has been maintained by tissue culture in bovine kidney cells for 100 serial transfers. It produced cytopathogenic effects. Temperature elevations that lasted from two to six days were induced within 18 to 36 hours when virus was given intratracheally, and within 72 to 120 hours when given intranasally. The virus was transferred from infected calves to others by contact.

In addition to increased temperature, infected calves showed salivation, lacrimation, inappetence, and depression. The calves developed neutralizing antibodies and, after recovery, were immune to subsequent inoculation of virus. In calves inoculated intranasally, the virus concentration was greatest in nasal exudate. Calves inoculated intratracheally showed a high concentration initially in the lungs and later in nasal mucosa. After intranasal inoculation, virus appeared in nasal mucus the following day in low concentrations and gradually increased, reaching its highest titer five days after inoculation. Virus was not recovered in nasal exudate 22 to 60 days after inoculation.

Neutralizing antibodies were found in some cattle from dairy herds in New York and also in calf serum collected in 1941 in New Jersey. Therefore, it was considered probable that infectious bovine rhinotracheitis occurs in cattle in the eastern United States and has been present for a long time.—[*J. H. Gillespie, K. M. Lee, and J. A. Baker: Infectious Bovine Rhinotracheitis. Am. J. Vet. Res., 18, (July, 1957): 530-535.*]

A California hog feeder was fined $500 and placed under a three-year probation for failure to comply with a law effective Jan. 1, 1956, that requires all commercial garbage fed to hogs to be sterilized at a boiling temperature for 30 minutes prior to feeding.—*Nat'l. Hog Farmer (Sept., 1957): 12.*

Lymphatic Leukemia in a Hog Following Atomic Exposure to *Gamma* Radiation—A Case Report

Lt. Col. BERNARD F. TRUM, V. C., U.S. Army, and
Lt. Col. WALTER T. CARLL, U.S. Army (Ret.)

Oak Ridge, Tennessee

ALTHOUGH LEUKEMIA in swine has been observed since 1865,[5] we believe this to be the first case clinically diagnosed and reported. A case was diagnosed at necropsy in 1926.[1] The incidence of swine leukemia in Europe over a 20-year period was reported, in 1927, as 3 out of 1.5 million necropsies.[5] In 1932, the incidence in the United States, according to the U.S. BAI,[6] was 1:220,000 (204,870,269 swine examined). However, later (1955) reports[11] gave an incidence of leukemia or lymphoma of 1:70,000 (classified together, out of 57,055,438 inspected swine).

An increase of leukemia following irradiation was reported in rats, in 1930,[9] and in mice, in 1936.[7] Primary lymphatic leukemia occurred in guinea pigs that were exposed to 8.8 r daily.[2]

In 1942, 24 cases of leukemia were reported in people working with ionizing radiation.[4] Articles concerning the high rate of leukemia among physicians were published in 1944[8] and 1947.[3,10] It was pointed out that among radiologists and nonradiologists in the medical profession there was an eightfold preferential of leukemia among radiologists, although some nonradiologists were also using x-ray equipment.[12]

Because of the associated radiation exposure and a primary lymphatic leukemia in an animal larger than man, it is believed that the case reported here is not only of historical importance in veterinary science but may be of interest to investigators of neoplastic pathology.

HISTORY

One of a group of 56 Yorkshire-Hampshire hogs died on the 196th day of an experiment during which all received, until death, 50 r daily of *gamma* radiation from Co-60 given at a rate of about 30 r per hour. The exposure device has been described.[13] The leukemic hog (51-4) was the thirty-sixth of the group to die. All others died within a year,* of a chronic irradiation sickness. No other leukemia was observed.

This hog had previously received 425 r of *gamma* radiation given continuously at about 1.5 r per hour and had been allowed a recuperation period of three months before the subsequent irradiation program was initiated. The total accumulated dose was 10,225 r measured in air.

The hog weighed 140 lb. when the experiment was initiated, 320 lb. when the chronic daily exposures began, 540 lb. one week before death, and 510 lb. at the time of death.

Two weeks before death, lethargy and a retrogression of general condition was observed, and a short walk usually produced exhaustion. No glandular enlargements were seen, although a splenic rigidity and enlargement could be palpated through the abdominal wall during the last week of life.

HEMATOLOGY

A tabulation of blood cytology is presented (table 1). Fourteen days before death, an increase in white blood count and a differential count, indicating 93 per cent lymphocytes, suggested a malignant lymphoma. The count increased to 345,000 leukocytes per milliliter of blood, with more than 99 per cent lymphocytes on the day of death. About 75 per cent of the lymphocytes were large.

Moderate anemia and severe thrombocytopenia were present just before death.

NECROPSY

Necropsy was performed on the barrow three hours after death. The carcass was well nourished and fat. There were no visible cutaneous lesions or discharges from body orifices. Swellings or enlargements of external lymph nodes were not seen, nor could any be palpated.

On primary incision, the cervical nodes were found enlarged and swollen. Single nodes weighed 12 to 25 Gm. Pleural and peritoneal lymph nodes were of apparently normal size. Nodes were yellow-white with purplish margins. Inflammation and pus were present in the tonsillar region.

Lungs.—The left lung weighed 780 Gm.

From the University of Tennessee, Atomic Energy Commission, Agricultural Research Laboratory, Oak Ridge.

The authors thank Fannie H. Cross for technical assistance.

*To be reported in Radiation Research.

TABLE I—Blood Cytology of Pig 51-4

Date		Hematocrit	Thrombocytes x 10^3	Leukocytes x 10^3	Lymphocytes (%)	Neutrophils (%)	Other (%)
Month	Day						
Preirradiation		39.0	490	21.4	65	21	14
July (24th day)	5	38.5	280	12.7	48	40	12
August	8	40.5	210	5.4	43	43	14
September	6	41.5	245	5.7	56	25	19
October	10	39.0	400	6.9	52	36	12
November	7	44.5	370	8.3	45	30	25
December	5	42.0	220	12.3	77	10	13
	12	38.0	90	43.5	93	—	—
	22	28.5*	50	181.8	99	—	—
	26	—	—	341.0	99	—	—
	27	(died)	—	—	—	—	—

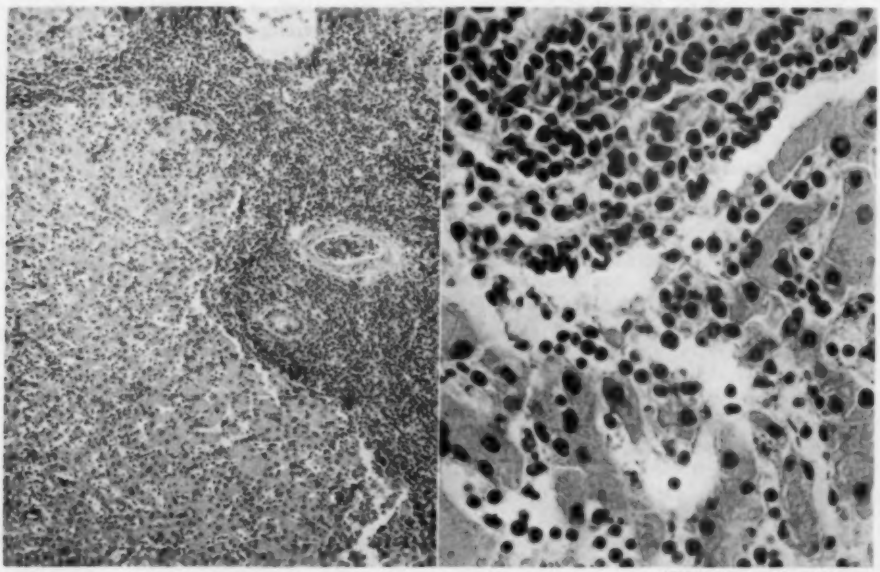
*The normal range of erythrocyte count for this group of pigs was 7.5-8 million per milliliter. The r.b.c. count of pig 51-4, on December 22, was 5.26 million per milliliter.

and the right lung 915 Gm. The pleural surfaces of all lobes had many elevated, dark purplish areas 1.0 to 1.5 cm. in diameter. Many white, hard nodules (1.0-10.0 mm.) were scattered throughout the pleural surface. On incision, it was seen that they contained a purulent material. The purple areas were bloody and engorged. The trachea and the bronchial tree contained a pink frothy fluid. Lymph nodes showed no gross changes.

Heart.—The heart weighed 500 Gm. It was of normal shape and symmetry. Petechiae were present on the edges of the left auricle and clotted blood was present in both the left and right compartments of the heart. Several hemorrhagic areas were under the endocardium of the left ventricle. Nothing unusual was observed in the major arteries or veins.

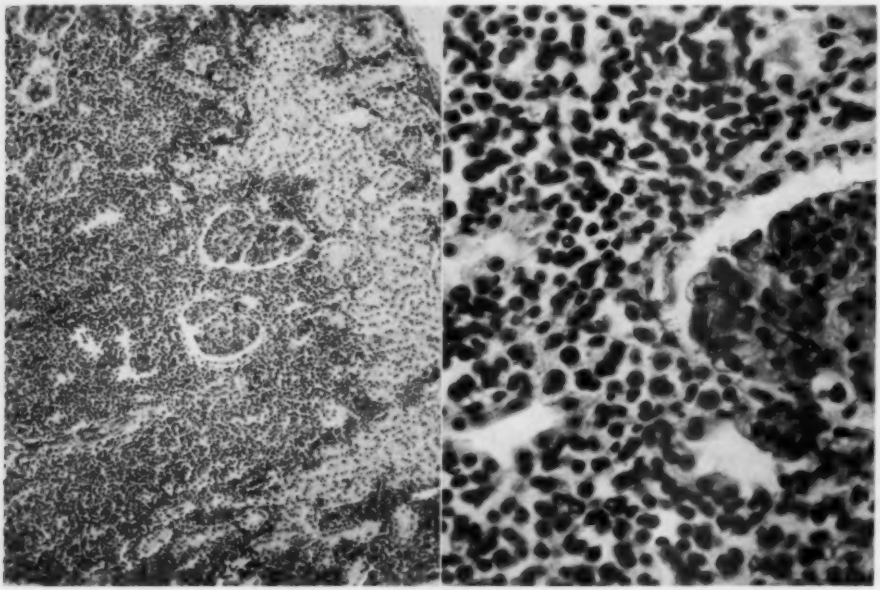
Spleen.—The spleen weighed 1,370 Gm. and measured 75 by 12 cm. and was 5 cm. thick in the middle. The edges were round and swollen. On the cut surface, it was diffusely dark purple and the architecture was distinct. The texture was considered friable.

Liver.—The liver, which weighed 4,470 Gm., was mottled with purple discolorations on a dark mahogany background. The



—*AFIP*-57-1642 *and* 57-1650

Fig. 1— The portal triads of the liver of the hog are especially prominent, due to invasion of lymphoid cells, and illustrate the condition found in other tissues of the body. x 80 (left); x 400 (right).



—*AFIP-57-1643 and 57-1648*

Fig. 2—The renal cortex of the hog shows intense infiltration by neoplastic cells. x 80 (left); x 400 (right).

lobules were exceptionally well defined and distinctly outlined by the interlobular connective tissue. On cut surface, the liver was friable and, in cross section, it resembled a nutmeg pattern. The bile duct and gallbladder appeared normal.

Adrenals.—The adrenals were light brown and showed bright red areas between the cortex and medulla.

Thyroid.—The thyroid weighed 20 Gm. and was purple. Many dark areas were seen on the cut surface.

Pancreas.—The pancreas was translucent and gelatinous.

Gastrointestinal Tract.—Nothing unique or abnormal was observed in the small intestine. The cecum was thickened (1-2 cm.) throughout. The mucosa presented a hobnailed appearance with well defined circumscribed hard white nodules with well indurated ulcerations.

Kidneys.—The right and left kidneys weighed 315 Gm. each. Many red areas were visible externally and on the cut surface many hemorrhagic areas and streaks were seen in the cortex. The streaks radiated from the medulla to the periphery of the cortex.

Brain.—The brain weighed 95 Gm. and nothing unusual was seen. The pituitary appeared slightly enlarged and the blood vessels were engorged.

COMMENTS

The most notable changes seen at necropsy were: the inflamed and pustular tonsillar area, the huge spleen (11 x larger than others of the group; 7 x on a Gm./kg. basis), the large (1.5 x normal of group on Gm./kg. basis) and beautifully patterned liver due to excessive leukocytic infiltration along normal interlobular demarcations of pig liver, and the "hobnail" mucosa of the cecum. It may be restated that, except for the cervical lymph nodes, there were no obviously enlarged nodes in the body.

MICROSCOPIC EXAMINATION

Lymph Nodes.—Severe infiltration by neoplastic cells obliterated much of the architecture of the lymph nodes. The cells were individually discrete, uniform, larger than normal lymphocytes, and round with scant cytoplasm. The nuclei were hyperchromatic and coarsely stippled. The capillary bed was engorged with erythrocytes and tumor cells. The capsule and trabeculae

were infiltrated by tumor cells. Reactive centers could not be distinguished.

Tonsils.—The tonsils were infiltrated and their architecture obliterated by neoplastic cells. The adjacent pharyngeal submucosa, muscularis mucosa, salivary glands, fat, and connective tissue had large numbers of tumor cells.

Lungs.—Alveolar edema, bacteria, and thrombi in the veins were seen in the lungs. The blood vessels had many neoplastic cells.

Spleen.—Splenic follicles were largely replaced by neoplastic cells. The trabeculae were recognizable and sinusoids were engorged with erythrocytes and tumor cells.

Liver.—Large numbers of tumor cells infiltrated the portal triads and interlobular septa, causing them to be especially wide and prominent at low magnification. Tumor cells were numerous in the hepatic sinusoids although they rarely formed nodules or displaced liver parenchyma (fig. 1).

Adrenals.—The medulla was densely infiltrated with neoplastic lymphoid cells while the adjacent cortex had fewer. A focal nodular hyperplasia of cortical cells was observed.

Pancreas.—In the pancreas, leukemia cells were contained in the vascular system.

Small Intestine.—Lymphatic tissue was replaced with leukemic cells. Areas of hemorrhages were in the mucosa, with free blood in the lumen.

Large Intestine.—In the cecum, there was widespread infiltration of lymphocytic cells throughout all the structures from the serosa to the mucosa. Fatty infiltration was present in the submucosa. In some areas, there was karyorrhexis of the leukemic cells with masses of nuclear debris. The mucosa had undergone advanced degenerative changes, the staining qualities had been impaired with a resultant ghost-like appearance of the remaining visible mucosa. Superimposed on the degenerative mucosa was a necrotic mass of tissue containing cellular debris of old hemorrhage, fibrin strands, remnants of leukemic cell infiltrations and extension.

Kidneys.—The renal cortex was intensely infiltrated by tumor cells and, in some areas, hemorrhage. The neoplastic cells isolated the renal tubules from one another in most of the cortex. In some cases, compression and obliteration of renal tubules was seen. Colonies of bacteria, not associated with an inflammatory reaction, were seen in the cortex (fig. 2).

Brain.—The lymphocytic cells were contained within the vascular system. A few small hemorrhagic areas containing lymphocytes were visible.

Pituitary.—Areas of leukocytic infiltration in the parenchyma were observed.

COMMENTS

No attempt was made to name the primary site of origin, although the cecal lesion was most developed and showed major degenerative changes.

Microscopic study revealed massive lymphocytic infiltrations in all tissues with many immature blastic cell forms in the blood vessels. The cells seen in the metastatic infiltrations of tissue examined were large, round lymphoid cells or less mature lymphocytes of uniform size and staining quality.

Microscopic vascular permeability as indicated by hemorrhage within tissue may have been associated with the thrombocytopenia.

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Behind the Scenes in Television at Cleveland

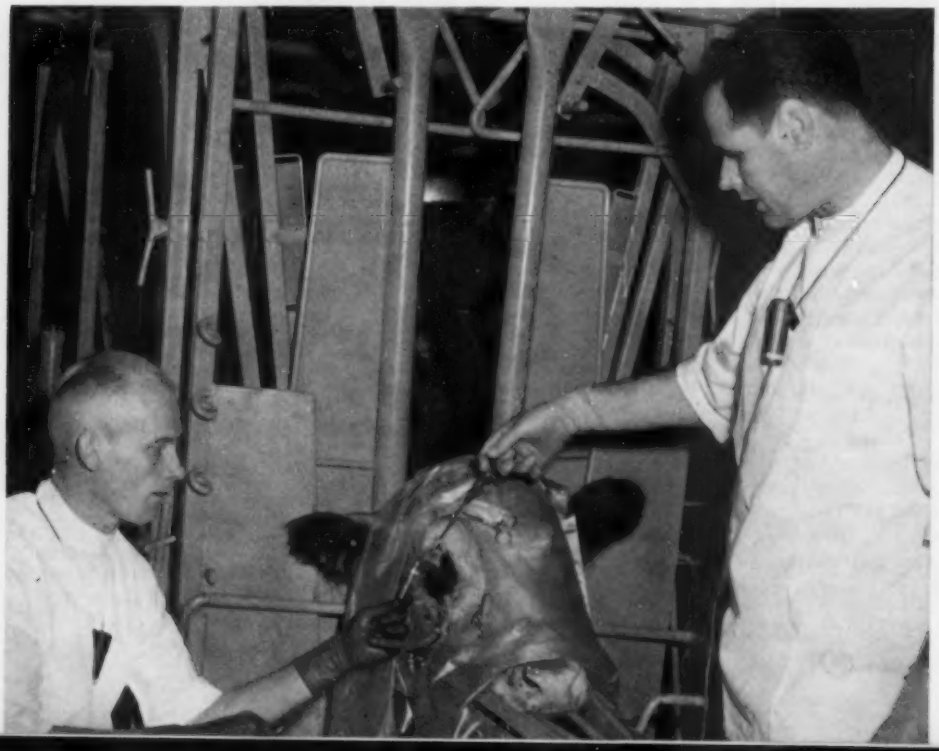
Dr. R. E. McKinley, Erie, Pa., demonstrated a new sling for "downer" cows.

Veterinarians who have seen only the television programs as they come over the viewers in the meeting rooms may be interested in the pictures on these pages, illustrating the complicated equipment, including cameras, cables, viewers, and other technical electronics that are needed to produce the closed circuit television at AVMA annual meetings.

An enormous amount of planning by the section officers and the local committee goes into the televised portions of the scientific programs each year, which are made possible by the Pitman-Moore Company, Indianapolis, Ind., in cooperation with Radio Corporation of America.

The smooth presentation of the televised program was due to the efforts of Dr. Les Fisher, Berwyn, Ill., the TV coordinator.

Drs. R. L. Lundvall (left), Ames, Iowa, and H. D. Simpson, Pearl River, N. Y., explained an improved technique for enucleation of the bovine eye.



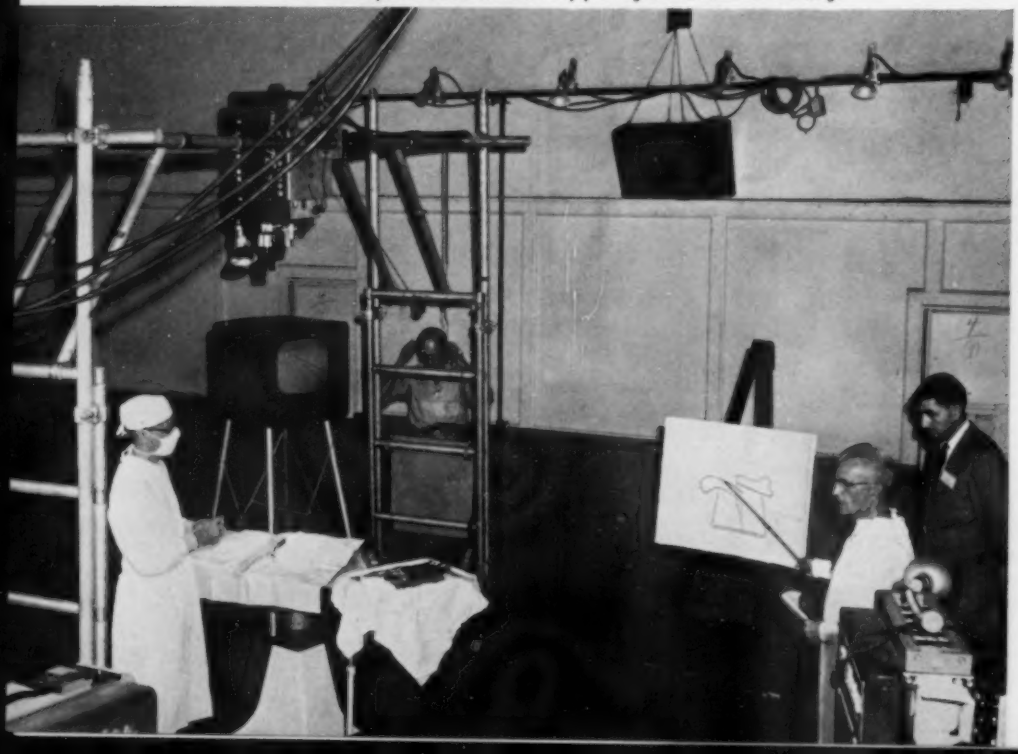
Dr. H. H. Berrier (left), Columbia, Mo., demonstrating diagnostic aids.

Drs. M. L. Willen (left), Canton, Ohio, and L. E. Green, Akron, Ohio, showed practice aids, techniques, and gadgets to the Section on Small Animals. →

Dr. W. F. Hoffman, Pittsburg, demonstrating new surgical equipment. ↓

Dr. J. E. Altman (right), Brooklyn, N.Y., explains techniques for the handling and treatment of caged birds. Dr. C. E. De Camp of Scarsdale, N. Y., looks on. Dr. L. E. Fisher, Berwyn, Ill., television coordinator, is at left.

Drs. R. L. Leighton (left), the surgeon, and Dr. C. E. De Camp, the narrator, both of New York City, illustrated permanent intramedullary pinning of the femur in the dog.

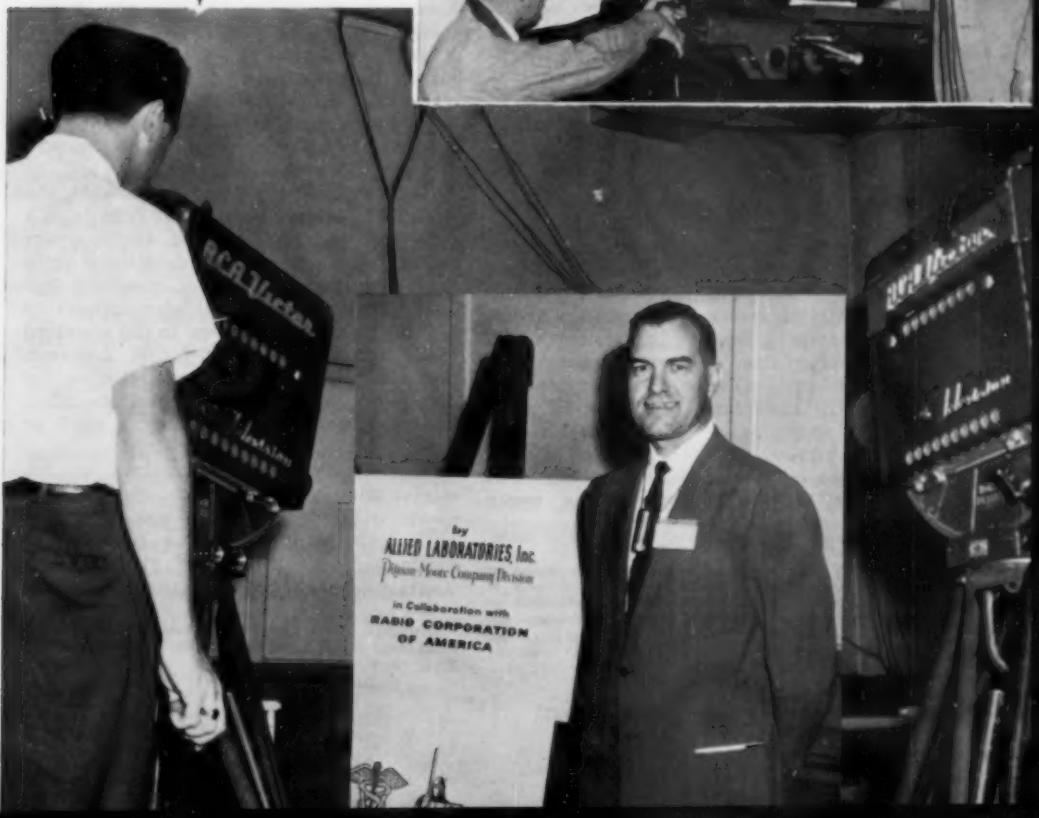


Dr. E. P. Leonard (left), Ithaca, N.Y., demonstrated the techniques of suturing.

Dr. D. L. Proctor, Jr. (left), Lexington, Ky., demonstrated the intra-articular and intrabursal injection of corticosteroids.

Dr. Stanley Ferguson (left, back) demonstrated the amputation of the bovine claw at the pedal joint. Dr. Edwin Krueger, Evansville, Wis., assisted and Dr. Rolland Anderson, Elkhorn, Wis., was the narrator.

President W. W. Armistead addressed the Closing Session of the Ninety-Fourth Annual Meeting over closed circuit television at Cleveland.



Surgery and Obstetrics
and Problems of Breeding

Canine Glaucoma. IV. Medical and Surgical Treatment*

W. G. MAGRANE, D.V.M., M.Sc. (Med.)

Mishawaka, Indiana

THE TREATMENT of glaucoma, whether it be primary or secondary, is both medical and surgical. In the dog, however, medical treatment as the sole means of controlling tension has proved unsatisfactory. It is not because medical treatment fails in every instance to control tension but because, even though drugs may be effective, pet owners become lax in treatment and fail to recognize the return of tension increases. Miotics often must be used continuously, two or more times a day, in order to maintain a normal tension, and failure to do so results in further loss of vision and eventual blindness. Also, most owners are reluctant to assume the responsibility of daily care and observation necessary over a period of years. It has been our experience that, in every instance where control depended upon drugs, eventually those drugs failed and surgery was necessary. Usually, vision had been progressively lost by that time.

We now prefer a surgical procedure in all cases of glaucoma, but use medical treatment to normalize the tension whenever possible, both as an emergency measure in acute congestive attacks and to facilitate the surgical procedures. Medical treatment is also needed, in some instances, to augment surgical procedures.

MEDICAL TREATMENT

The nonoperative treatment for primary glaucoma consists primarily of the use of miotics. If effective in constricting the pupil, miotics reduce intraocular tension by (1) opening up the cornea-iris angle, (2) opening the posterior venous channels, (3) constricting the blood supply into the ciliary body, and (4) allowing more surface area of the iris to absorb the aqueous humor.

The two miotics most commonly used in the past, eserine and pilocarpine, never satisfactorily controlled acute congestive attacks in the dog. It was not until the advent of di-isopropyl fluorophosphate (DFP)† that some hope appeared for medical control of primary glaucoma in these animals.[1-6] It was used in 0.1 per cent solution; no ill effects were ever noticed following its use. In many instances, it tended to lower, if not normalize, tension. However, with tensions of 60 to 80 mm. Hg., it often had no effect unless it was combined with another drug, acetazoleamide 2-acetylamino-1, 3, 4, thiadiazole-5, sulfonamide (Diamox§), a powerful carbonic anhydrase inhibitor. The initial clinical application of this latter drug was as a diuretic, in which field it has achieved widespread acceptance because of its effectiveness and low toxicity. It acts in the eye by inhibiting the formation of aqueous humor. It has no effect on the outflow and so should be used in combination with miotics. It is synergistic with miotics since they act on the two different mechanisms producing increased intraocular pressure, secretion of aqueous fluid, and decreased outflow. By lowering the intraocular tension through decreased secretion, miosis is achieved more readily by miotics; thus the angle is unblocked and drainage can occur.

Grant and Trotter[9] concluded that the latter drug's value seems most definite as a supplement to the standard miotic drugs in the relief of glaucoma caused by acute angle-closure. In attacks of glaucoma secondary to iridocyclitis, it also appears to be helpful as a supplement to the standard local medications. They found, however, that while the drug has been effective for months in some cases, in others the effect has been maintained for only a week or two.

Becker[2] recorded observations in administration of this drug to over 300 patients with glaucoma and was impressed with its effectiveness as a preoperative medication in all types of glaucoma. Its use makes possible an operation on a softer,

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*Abstracts from the thesis accepted by the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Master of Medical Science in ophthalmology.

This work was made possible by a fellowship granted by the Research Council of the American Veterinary Medical Association.

†Floropryl, Merck Sharp and Dohme, Philadelphia, Pa.

§Diamox is produced by Lederle Laboratories, Pearl River, N. Y.

less inflamed eye with less likelihood of complications.

In the dog, its principal undesirable side effects in this study were signs of nausea and vomiting. This occurred in some instances with doses as little as 75 mg. Other dogs became drowsy, and increased urination was not uncommon. It was necessary, by means of trial and error, to establish the dosage for the dog of average weight (20-30 lb.), since the majority of those afflicted with glaucoma (Cocker Spaniel and Wire-Haired Terrier) are in this weight range. A standard dose now consists of 125 mg. every eight hours for three doses, followed by 125 mg. every 12 hours. Side effects have been reduced, and effectiveness maintained with this dosage.

When the two drugs are used in combination, 1 drop is instilled in the affected eye every hour for six instillations. It is then used two or three times a day until surgery is performed. The importance of this combination of drugs in normalizing tension, thus preventing further visual loss and preparing a softer eye for surgery, can not be overestimated. Either alone or in combination, DFP is never depended upon to effect permanent normalization of tension for reasons already expressed, except in certain instances. If surgery is rejected or the animal is very old, the combination may be used as long as it proves effective or is without undesirable side effects.

The retrobulbar injection of pure grain alcohol has been advocated in blind, painful eyes for relief of pain and for a possible reduction in tension; 1.5 cc. of pure grain alcohol was injected retrobulbarly in 2 dogs with absolute glaucoma. In 1, a severe reaction followed the injection and within ten days a large central ulcer developed and the eye was extremely sensitive and irritated. The final outcome is unknown. Results of retrobulbar injection in the second dog, which showed signs of pain in the affected eye, were good. Tension remained normal during an observation period of several months, and it was learned by correspondence one year later that, although the eye remained enlarged and the episcleral vessels seemed distended, there had been no evidence of pain in the eye since the injection. This procedure may have merit in absolute glaucoma when surgical procedures are rejected or fail. For the practitioner who anticipates enucleation of a blind, glaucomatous eye, there is nothing to lose and everything to gain by attempting the procedure. It may be repeated at intervals, if necessary, and is easily performed.

Under short-duration barbiturate anesthesia, a 1¼- to 1½-inch, 22-gauge needle is inserted to a point just behind the posterior surface of the globe. Several approaches may be selected but the writer prefers to insert the needle through the conjunctiva and ventral to the globe along the inferior orbital ridge between the 3 and 6 o'clock position; or dorsally between 9 and 12 o'clock, following, by feel, the cartilaginous orbit. The globe should be fixed in the opposite quadrant with fixation forceps. After injection, the needle is withdrawn and a slight rotary pressure is exerted on the globe through the lids in order to promote thorough diffusion within the cone.

SURGICAL TREATMENT

As pointed out previously, the treatment of choice for glaucoma, regardless of type, is practically always surgical. There is, however, no one surgical procedure to care for all glaucomas. A careful examination and an accurate classification is necessary in order to determine the type of procedure that is best suited for each case.

The prime objective in the surgical treatment of glaucoma is to secure a lower tension and preserve vision; and second, if the condition be absolute, to preserve the integrity of the globe—in other words, to avoid enucleation. Since so many glaucomas in the dog are in the absolute stage before the dog is presented, pet owners are usually pleased if, through surgery, the dog's eye is rendered painless and the buphthalmos is checked. They much prefer this to enucleation and closing of the orbit, which is almost inevitable if the buphthalmos is allowed to progress. For these reasons, surgery of the blind eye is practical in the dog.

It has been said that enucleation of a glaucomatous eye in the dog will prevent glaucoma in the second eye. This has not been our experience. In the past, attacks of glaucoma in the second eye, following enucleation of the first, occurred in several instances within a matter of days and often within months.

The operations performed in the dog in this series may be classified as follows: iridectomy (peripheral, basal, prophylactic); filtering cicatrix (iridencleisis, Elliot trephine); cyclodiathermy; and lens ablation (intracapsular, extracapsular).

PREOPERATIVE PREPARATION (ALL PROCEDURES)

Twenty-four hours prior to surgery, the hair around the eye is clipped with electric clippers or scissors. With blunt-pointed scissors, the blades of which have been anointed with either water or mineral oil, the lashes of the upper lids are cut. It is important that all hair and lashes be removed from the operative scene, not only for aseptic reasons, but because a loose lash is difficult to remove from the interior of the eye and, if left, it will produce most unfavorable consequences. The lids may be cleansed with a surgical detergent and water, but extreme care must be exercised and the eye protected with ointment or oil. Trinitrotoluene in a cleansing agent (Phisohex) was reported to quickly penetrate the canine cornea, producing a deep keratitis and epithelial loss, and hydropic swelling and sloughing of the corneal endothelium.[1] We have had this occur on several occasions and lost one eye as a result. We, therefore, massage antibiotic ointment thoroughly into the lids and instill it in the eye several times before surgery. This seems adequate and detergent cleansing is now reserved for eyelids which are obviously unclean.

Intraocular operative procedures must be undertaken in the most aseptic manner. The operator should wear cap, mask, gown, and gloves. In addition to standard draping of the operative area, a rubber dental dam should be used to block out all of the animal but the eye. A 4- by 4-inch strip of dental dam is included in the pack. A slit corresponding to the length of the palpebral fissure is made with knife or scissors, then the dam placed over the eye and the speculum inserted. In this manner, the dam rolls over both upper and lower lids, exposing the eye and its membranes only.

The following operations will not be described in minute detail since many excellent texts on ocular surgery give this information. In discussing results of various operative procedures, "permanent" means that the animal was observed for six months or longer, and that the tension remained normal. This would correspond to a period of two years or longer in man, which is adequate for evaluating a successful procedure.

IRIDECTOMY

This operation is indicated within the first day of an attack of primary glaucoma. If postponed, it is apt to fail because peripheral anterior synechias will have begun to develop. Both peripheral and basal iridectomies were performed, peripheral meaning that the base or iris root was not included in the excision. Our series is too small to properly compare the effectiveness of these two procedures.

In one acute congestive attack, a basal iridectomy afforded permanent relief. In five eyes afflicted with secondary glaucoma, subsequent to an iris *bombé*, either a basal or peripheral iridectomy effectively normalized tension. In one instance of luxated lens, an iridectomy alone gave permanent relief but it is certainly not the operation of choice in secondary glaucomas of this nature. However, a peripheral iridectomy is now always included in the lens ablation operation.

How iridectomy affords tension relief is not exactly known. There is evidence to support the mechanical theory that it tends to deepen the periphery of the anterior chamber angle and opens a blocked angle. Some believe that iridectomy acts by way of operative trauma and that the circulatory system is more or less permanently modified through nervous mechanisms. In iris *bombé*, most authorities agree that tension is normalized through establishment of circulation of aqueous fluid from the posterior to the anterior chamber.

Since second eye involvement (acute congestive glaucoma) is common in man and dog, and also since the first eye is often blind, we now try to persuade the owner to have the other eye operated on. We have performed a prophylactic iridectomy on the normal eye of 5 dogs at the same time the affected eye was operated on.

Prophylactic peripheral iridotomy in the unaffected eye has been recommended in all cases;[5] in all young and middle-aged patients with acute glaucoma;[2] and as an alternative to continuous therapy with miotics in acute congestive glaucoma due

to shallow chamber angle.[3] An iris inclusion operation is recommended as a method of achieving the same end.[4]

FILTERING CICATRIX

Filtering cicatrix operations have as their object the formation of a permanent filtering cicatrix at or near the sclerocorneal junction through which the excess of aqueous fluid filters out under the conjunctiva. While this has been generally assumed to be the mechanism whereby tension is reduced, considerable evidence to the contrary has been produced.

After an exhaustive study,[6] it was concluded that the persistence of a small area of conjunctival edema after iridencleisis does not definitely demonstrate that the scar is the site of a real filtration. Scars are often flat and seem to exclude the possibility of any filtration, and yet the tension reduction is excellent. Iridencleisis may lower and normalize the ocular tension by inducing complex neurovascular modifications at the level of the anterior uvea.[6]

In the majority of cases in one series of successful iridencleisis operations, the resistance to aqueous fluid outflow was brought down to normal range and the operative area did not show any signs of fluid filtration into subconjunctival tissue spaces.[7] These findings suggest that the action of the iridencleisis consists in enhancing or reestablishing the function of the normal outflow channels. Only in about one fourth of those cases did a permanent bleb form.

Following iridencleisis, we have seen functional blebs remain a long time, but the scars usually became flat eventually. In those with flattened scars, the tension remained normal in some but became elevated again in others. However, in all in which the bleb appeared functional, the tension remained normal.

Of the 18 iridencleisis operations we performed in primary glaucomas, ten gave permanent relief with 2 of the 10 dogs needing miotics in addition. In the other 8, it was necessary either to make an additional trephine opening, or to resort to cyclodiathermy or lens ablation in order to normalize tension.

In the secondary group, 13 iridencleisis operations were performed, with permanent relief in six. Only 1 of 5 dogs with lens displacement responded to an iridencleisis. In 1 with iris *bombé* and in 2 with uveitis, the relief was permanent. Of 3 dogs in which injury and adhesions were believed to be the cause of the secondary glaucoma, the pressure in 2 was normalized by iridencleisis.

The Operation.—Many variations of iridencleisis or iris inclusion operations are described. Our technique has been varied to include: (1) incarceration of one tag of iris following a split of the iris from base to sphincter; (2) incarceration of both tags following the split; (3) excision of a wedge of iris before incarceration of a tag; (4) suturing the tag to the episcleral tissue; (5) incarceration of a tag through a trephine hole (sclerectomy); and (6) application of Gelfilm* to the incarcerated tag.

The principal steps in the operation are:

1) The conjunctival flap is prepared, starting about 8 mm. from the limbus, undermining the conjunctiva so that the sclera is bared. The undermining should extend to the limbus in the central portion of the flap only.

2) The incision, 4 to 5 mm., through the sclera into the anterior chamber is made with a No. 11 blade or keratome about 1 mm. behind the limbus.

3) The iris is allowed to prolapse or is grasped with iris forceps or a dull iris hook and brought to the outside. Holding the iris with forceps on each side, an incision is made from the base through the sphincter muscle and free border and one or both tags are pulled to the edge of the scleral incision where it is incarcerated. If only one tag is used, the second is allowed to return to its position in the anterior chamber.

4) The conjunctival flap is replaced over the incarcerated tag and sutured in place with 4-0 chromic catgut. A drop of atropine solution (2%) is instilled and the eye bandaged.

In order to secure permanent filtration from the anterior chamber to the immediate subconjunctival spaces, a sterile, nonantigenic, absorbable film made from pig gelatin has been utilized.[8] When dry, it looks and feels like cellophane, but when wet it assumes a soft rubbery consistency which molds well to any body surface. Its absorption requires from one to six months,

*Gelfilm—Upjohn Company, Kalamazoo, Mich.

depending upon the tissue in which it is implanted, and an opening or "envelope" remains between the conjunctiva and sclera.

In the last 4 cases of primary glaucoma treated by iridencleisis, a piece of gelatin measuring approximately 5 by 8 mm. was moistened in sterile saline solution and placed over the iris tag parallel to the limbus before replacement and closure of the conjunctival flap. During the year or more which had elapsed since surgery on these dogs, there had been no reaction from the procedure, and the tensions remained normal. A filtering bleb is still in evidence.

When the iris becomes atrophic or degenerated, an iridencleisis is more likely than not to fail. Trephination should then be considered instead of the iris incarceration. Again, there is a difference of opinion as to whether the results produced by trephining are due to fistulization or other factors.[9,10]

Trephination was performed in three eyes. In two, additional miotics were needed following surgery. A third eye was subjected to a combined trephination and iridencleisis with permanent relief.

Briefly, in trephining, a conjunctival flap is first prepared, extending the dissection to include the corneal epithelium for several millimeters in the center of the flap. A 2- to 3-mm. trephine is then used to remove a disc which includes one half cornea and one half sclera. An iridectomy or iridotomy should be combined with the trephination. The conjunctival flap is replaced and sutured in the manner described for iridencleisis.

CYCLODIATHERMY

By definition, cyclodiathermy is the application of diathermic current to the ciliary body through the sclera by means of penetrating or nonpenetrating electrodes.

The exact mechanism of action of cyclodiathermy in lowering intraocular pressure is not completely understood.[11] Whether it acts primarily to destroy the secretory function of the ciliary body by interfering with its vascular supply or whether it serves to stimulate the formation of a new anastomotic group of vessels and thus effect outflow of aqueous fluid has not been established. Some[12] advocate its use primarily when all other procedures have failed, while others are of the opinion that the procedure should be reserved for cases in which filtering operations are contraindicated, or in which outflow following a filtering procedure is mildly inadequate.

We used cyclodiathermy on eight eyes, usually after one or more procedures had already been tried and failed to normalize tension. In three of four eyes which were in the primary classification and were blind, the tension was rendered normal permanently; in the fourth, a miotic was needed in addition. Four eyes were in the secondary classification. Cyclodiathermy failed to reduce tension in one eye with chronic uveitis and iris atrophy. However, in an aphakic eye (postcataract extraction), relief was permanent. In another eye with injury and adhesions, the result was good, and in the fourth (subluxated lens), the procedure reduced but did not normalize tension.

The Operation.—While varying techniques for applying the electrodes have been described, we employed the same one[12] in all eight eyes. A Miller electrosurgical unit* was used. The technique follows:

1) The eye is draped and fixed below with fixation forceps so that the upper portion of the globe is well exposed and the bulbar conjunctiva taut.

2) A 1.5-mm. electrode is applied through the conjunctiva and into the sclera, 5 to 6 mm. behind the upper limbus, and at 8 to 10 points evenly spaced to cover the upper half of the globe. A millimeter rule may be used for the measurement.

3) With the electrode set at 1 (to produce coagulation), it is held in place for ten seconds at each application.

4) Atropine solution (2%) and an antibiotic are instilled and the eye is bandaged for two days.

Occasionally, there will be a loss of fluid from some of the punctures, which is desirable. A moderate chemosis (edema of the conjunctiva) will be in evidence for several days following the procedure. This is alleviated by use of hydrocortisone drops or ointment. A secondary rise in tension may follow the operation but this soon subsides. We are enthusiastic about cyclodiathermy for relief of absolute glaucoma in the dog. It is easily performed and may be repeated as needed. Buphthalmia subsides after several weeks, and some eyes even appear to return to normal size.

LENS ABLATION

Lens extraction is recommended[14] in glaucoma: (1) due to swelling or intumescence of the lens; (2) when it is secondary

*Miller Surgical Electro-surgical Equipment.

to lens-induced uveitis, e.g., capsule rupture in hypermature cataracts; (3) due to subluxation or luxation of the lens; (4) when it is malignant, and (5) when due to acute iris block of an advanced nature when accompanied by a very shallow anterior chamber and a high tension which does not respond to medication. Malignant glaucoma was defined[14] as one in which surgical intervention is promptly followed by a flat anterior chamber and an elevated tension, both of which are unresponsive to further measures.

The most frequent operation to be performed in canine glaucoma is removal of the lens, the displacement of which is responsible for the secondary tension rise.

A total of 39 lenses were removed by either the intracapsular or extracapsular method. An analysis reveals the following statistics.

Secondary Glaucoma.—Permanent tension normalization occurred in 34 canine eyes after removal of the lenses, with good cosmetic results in 25 (73.5%). One lens removal was combined with an iridencleisis and 1 dog required a miotic afterward. Extracapsular extractions were performed in three of the 25 eyes and intracapsular extractions in 22.

Of those listed as failures (9 of the 34), three terminated as phthisis bulbi (shrunken, fibrotic globe) after months or years, three were extracapsular, and three were intracapsular extractions.

Primary Glaucoma.—Two of the five lens removals were performed following an unsuccessful iridencleisis and three were initial procedures in cases of primary, absolute glaucoma. Two of the latter had normalized tension when discharged and tension was lowered, with the aid of a miotic, in the third.

We attempted intracapsular extraction in all instances of lens displacement. Occasionally, an extracapsular extraction resulted when, inadvertently, the capsule burst during extraction. This usually occurred when the lens was bound by adhesions. There were only three failures in 22 intracapsular extractions but three in six by the extracapsular method.

The Operation.—(1) Canthotomy.—The necessity of performing a canthotomy depends upon the amount of buphthalmos and exophthalmos. If the eye is well exposed, a canthotomy is unnecessary. If, however, more working space is desired, a few drops of adrenalin solution (1:1,000) are injected into the tissues of the lateral canthus and blunt surgical scissors are used to extend the lateral canthus for a distance of about 10 mm.

2) Retrobulbar Injection.—If additional anesthesia or further anterior prolapse of the globe is desired for ease in operation, 1.5 cc. of a 2 per cent novocaine solution is injected into the region of the muscle cone.

3) Preparation of the Flap.—Approximately 5 to 8 mm. from the upper limbus, the conjunctiva is incised around the upper half of the eye and dissected down to the limbus with tenotomy scissors.

4) Preliminary Groove.—Using a No. 15 blade, an incision, or groove, is made along the line of the contemplated section about 0.5 to 1.0 mm. posterior to the limbus and about one half the thickness of the sclera. One groove is made at the 11 o'clock and one at the 1 o'clock position.

5) Sutures Preplaced.—Using 6-0 silk on atraumatic eye needles, one suture is placed (fig. 1) in each of these grooves before the incision is completed, thus eliminating the hazard of manipulating the opened eye. The sutures (McLain type[15]) do not penetrate into the chambers of the eye. The needle, starting at the epithelial surface of the conjunctival flap, passes through it, then into the

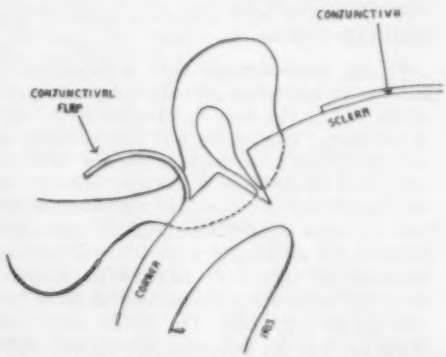

Fig. 1—A sketch showing how, in surgery for primary glaucoma of the dog, the suture is inserted before the incision into the anterior chamber of the eye is completed.

sclera posterior to the groove. It then passes anteriorly, in the scleral tissue, to cross the groove at its depth and emerges through the surface of the cornea. Each suture, where it is exposed in the groove, is picked up with a sharp iris hook to form a loop which is held away from the incision by a clamp, on each side, which is clamped to the upper and lower bars of the eye speculum.

6) Section.—With the eyeball fixed, by a forceps on each side, a keratome or No. 11 changeable knife blade is used to incise the sclera, in line with the grooves and at the 12 o'clock position, into the angle of the anterior chamber of the eye. The incision is then extended, with corneal

scissors, until it is adequate to avoid undue pressure in removing the lens. In lens displacement, the hyaloid membrane usually has been ruptured, consequently the vitreous will present itself upon opening the eye. To minimize loss of vitreous, there should be no pressure on the eye; thus, the speculum tension is reduced prior to enlarging the incision.

7) Iridectomy.—A peripheral iridectomy or iridotomy is performed and hemorrhage is controlled before the lens extraction is made.

8) Extraction.—A wire lens snare is used to encompass the posterior part of the lens, thus lifting the lens toward and through the incision. Iris scissors are used to cut clinging vitreous from the lens.

9) Sutures.—The sutures are drawn up and tied, the conjunctival flap is replaced and sutured with 4-0 chromic catgut on an atraumatic needle. In the dog, this suture may be continuous but should be tied on both ends. An air bubble is injected into the anterior chamber with a hypodermic syringe and lacrimal needle which is inserted between the conjunctival sutures. A drop of pilocarpine solution (2%) and an antibiotic is instilled, a single mattress suture is placed to close the lids, and a bandage is applied.

POSTOPERATIVE CARE (FOR ALL GLAUCOMA SURGICAL CASES)

Proper postoperative care is exceedingly important following all surgical procedures of the eye in the dog. Bandaging of the eye is necessary to cushion and immobilize the eye; to provide warmth as an aid to healing; and to prevent infection. The eye is bandaged with an eye pad* cut to size and held in place, first with strips of tape, and finally with an elastic tape.* Dressings are changed on alternate days with instillations of the same preparations as used following surgery. On the sixth day, the dressing and lid suture are removed. One of the antibiotic-hydrocortisone ophthalmic ointments is useful from the sixth to the fourteenth day, when the animal is discharged, to prevent neovascularization and to keep the eye "quiet." The corneoscleral sutures are removed on the tenth day. The conjunctival suture is left to absorb. Careful handling of the patient and freedom from excitement are important during hospitalization.

POSTOPERATIVE COMPLICATIONS

Postoperative complications are most apt to occur as a result of faulty technique, or the selection of an improper procedure for the type and stage of glaucoma presented. However, even when the technique is apparently faultless and the proper operation is selected, a certain percentage of operations are doomed to failure.

One author[16] reports a minimum of 15 per cent failure at the first operation, and at least 50 per cent at reoperation. He cautions against repeating a procedure which has failed once.

Glaucoma frequently leads to the formation of lens opacities which can not be avoided. Lens opacities and cataract, however, may be hastened by operative trauma, and care must be exercised to avoid touching the lens during filtering procedures.

Other postoperative complications include vitreous prolapse, uveitis, late infection, and phthisis bulbi. Clotted blood remaining in the anterior chamber following glaucoma procedures was a major complication. Hemorrhage will follow an iridectomy or iridencleisis, or even the preparation of the conjunctival flap and the scleral incision. Therefore, we now attempt to control all hemorrhage attending a step before proceeding with the operation.

SUMMARY

The treatment of glaucoma, whether it be primary or secondary, is both medical and surgical. Medical treatment can not be depended upon over a long period and is used primarily to normalize tension and prepare the eye for surgery. The drug, DFP (Floropryl), has proved to be the best of the miotics in the dog. Diamox is used *per os* and acts by diminishing production of aqueous humor (or fluid). A combination of the two drugs must be used for optimum results. A retrobulbar injection of pure grain alcohol may be tried in blind, painful eyes for relief of pain and for a possible reduction in tension.

There is no one surgical procedure suitable to care for all types of glaucomas. A careful examination of the glaucomatous eye and an accurate classification is necessary to determine the type of surgical procedure best suited for each case. The prime objective of surgery is to reduce tension and save vision but, when vision has already been lost, it is then important to preserve the integrity of the globe. Enucleation of a glaucomatous eye will not prevent glaucoma in the second eye.

An iridectomy is performed within the first day or so of an attack of primary

*Combined roll and Elasticon are produced by Johnson and Johnson, Chicago, Ill.

acute congestive glaucoma. A prophylactic iridectomy in the second eye is recommended. Iridencleisis is the operation of choice after the period for iridectomy is past. The use of a film of gelatin (Gelfilm) in the iridencleisis procedure seems to prolong the actual filtration.

A trephine operation is suggested when the iris becomes atrophic or degenerated. Cyclodiathermy may also be employed under these conditions. It is an especially useful and simple procedure to employ in absolute glaucoma.

In secondary glaucoma caused by subluxation or luxation of the lens, an intracapsular lens extraction will normalize tension in most cases. A lens ablation is also indicated when there is lenticular intumescence and when a hypermature lens causes glaucoma.

Postoperative care is exceedingly important and bandaging is recommended. Many postoperative complications can be avoided by improved technique, proper selection of operation, and control of hemorrhage.

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Use and Misuse of Acetazoleamide (Diamox).—In the treatment of glaucoma, acetazoleamide has a pressure-lowering effect due solely to the lessening of the rate of aqueous humor production. Its greatest field of usefulness is in secondary glaucoma—a rise in ocular tension secondary to some other eye disease. In patients with obstruction of the outflow, due to closure of the angle of the chamber, a simple surgical procedure will effect complete relief in the early stages but, in the late stages, all treatment may fail. The drug should never be used as a continuing treatment of angle closure glaucoma but it is useful as an immediate preoperative treatment.—*J.Am.M.A. (Sept. 7, 1957): 106.*

Surgical Wound Infections in Man.—Antibiotics have not solved the problem of postoperative wound infection. They have inspired a false confidence leading to a lack of basic aseptic surgical principles. Penicillin-resistant organisms are on the increase—one study showed 4.1 per cent

in 1953 and 7.3 per cent in 1956. Most wound infections at one hospital were due to hemolytic *Staphylococcus aureus*. They were present on the skin or in the noses of 49 per cent of the patients and 74 per cent of the house staff. When tests were made by holding agar plates 18 inches from the faces of nine medical personnel, the colony counts were higher when the person talked while wearing a double mask than with no mask when they were not talking, indicating that silence is more valuable than masking. Before an operation, two trays of instruments should be prepared and the surgeon should wear a double set of gloves; the superficial gloves and the first tray to be discarded when the "deep knife" is required.—*Am. J. Surg. (Sept., 1957)*: *398*.

An Extrathoracic Heart in a Parakeet

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On May 25, 1957, a female parakeet, 3 years old, was presented for treatment for a swelling about 1 inch in diameter located just anterior to the thoracic inlet. The swelling was soft and fluctuating with a firm core that could be moved freely within the swelling. The swelling was first noticed on May 11, 1957, at which time it was rather small. It had steadily grown in size up to the time of the examination. The bird was eating well with no regurgitation, although it had stopped talking and "just sat with ruffled feathers."

A diagnosis of either a fatty tumor or a cyst was made with recommendation for surgical removal of the mass. At the time of surgery, May 30, 1957, the swelling was slightly larger than when first examined.

Ether anesthesia was administered and the area plucked and prepared. A large vein, ⅛ inch in diameter, resembling a varicose vein, was present in the skin over the swelling. When the swelling was incised, about 5 cc. of straw-colored serum was removed along with three or four blood clots ranging in size from ¼ to ½ inch in diameter. The firm core could still be felt in the cavity so the incision was extended. However, upon closer examination, the mass was found to be the heart. The heart had a scarred surface where an epicardial capillary had been ruptured. The larger coronary vessels seemed to be intact and in good condition. The bird died from shock about 15 minutes after surgery.

Evidently, the bird had been born with the heart anterior to the clavicle and thoracic inlet, and had led a normal life until, flying against some object, it had ruptured the epicardial vessel, causing the large hematoma.

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Mink Breeding Peculiarities.—Ovulation in the mink is stimulated by copulation, which was found to average 64 minutes and was more than two hours in 10 per cent of the matings. To conserve male energy so that fewer males would be required, an experiment with interrupted mating was conducted in Sweden. When the male was removed after six minutes, 42 ova were recovered from 8 females; when separated after 12 or 24 minutes, 55 and 54 ova were recovered, respectively. After six minutes, too few spermatozoa were deposited, and the stimulation was insufficient to effect ovulation in all mature follicles.

Most females will mate several times during the season (early spring) and it is believed that ova released at early matings implant about the same time as those released at late matings. When females were bred once each by 2 males of different types, two-thirds of the kits could be identified as from the last mating when the interval was one day, and five-sixths of the kits as from the second mating when the interval was eight days or more.—*Am. Fur Breeder (Aug., 1957): 14.*

Preoperative Intestinal Antisepsis.—Erythromycin alone is not a satisfactory agent for preoperative preparation of the colon; erythromycin-neomycin is one of the most effective combinations yet studied for intestinal antisepsis.—*Am. J. Surg. (Sept., 1957)*: *402*.

Bilateral Oviducts in Hens.—During artificial insemination, it was suspected that two oviducts were present in an inbred strain of Rhode Island Reds. Necropsies verified that a portion of the right oviduct had persisted in some of these hens but a right ovary was never found.—*Nature (July 13, 1957): 98.*

A Review of the Chemotherapy of Avian Viral and Bacterial Infections

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RECENT CHANGES in the methods of raising poultry have increased the problems in disease control. In Delaware and many other areas of the United States, the small family flock of 25 to 50 birds is a thing of the past. Single farms may house as many as 100,000 chickens and the floor space allowed to 1 bird may be less than three fourths of a square foot.

In the past, many veterinary practitioners have neglected poultry. However, with the present day mass methods of treatment and vaccination, as well as the extreme concentration of poultry in certain areas, poultry practice presents more of a challenge. The cost of poultry diseases is staggering. The data in table 1 were taken in a survey of the broiler flocks in the state of Delaware by the Department of Agricultural Economics at the University of Delaware.

Antibiotics and other medicaments are administered by numerous methods. They may be incorporated in the feed or drinking water or injected subcutaneously or intramuscularly. Streptomycin has been dispersed into the air as a dust. The success of such flock therapeutic measures depends upon each bird consuming or inhaling its share or quota of feed, water, or dust. These methods also serve for prevention, since the normal as well as the sick birds in the diseased flock are under treatment. In some cases, the value of such treatments is largely prophylactic rather than therapeutic. This is particularly true in infectious synovitis since it has been established that the medicament has no effect in birds already lame and crippled.

Antibiotics given as a single intramuscular or single oral dose produce detectable blood levels in one half hour, but these levels do not persist for more than 48 to 72 hours in most cases.[9] However, continuous administration in the feed at sufficiently high levels does maintain blood levels as long as the birds continue to consume the medicated feed.

TABLE 1—Broilers—Cost of Diseases in Delaware

		1954	1955
Average mortality (%)		9.37%	6.52%
Cost of medicine (% of total cost)		4.89%	4.46%
Cost of medicine per 1,000 broilers		$34.16	$30.17
Total cost of disease in one year:			
Loss of broilers	$4.5 million		
Cost of medicine	$2.5 million		
Total	$7.0 million		

CHEMOTHERAPY OF VIRAL DISEASES

Although the search for an antiviral agent has been intensified in recent years, there is no evidence that such medicament has been found except that a few antibiotics are effective against the large viruses. The most important of such treatments in the avian field is the chemotherapy of psittacosis (ornithosis) with chlortetracycline (Aureomycin*). At levels of 100, 200, 400, 600, and 800 Gm. per ton of feed, it was effective in preventing mortality from this disease.[11] Egg-propagated turkey ornithosis virus was inoculated into the tracheas of 3-week-old poults and treatments were started simultaneously. The birds given the 100 Gm. per ton level were visibly affected at the end of the 14-day observation period. The virus was isolated from them at this time by assay in mice and chicken embryos. No lesions were observed at necropsy in the poults of groups receiving 200, 400, 600, and 800 Gm. per ton. The virus was isolated from 8 per cent of the birds given 200 Gm. per ton, but not from those given more than 200 Gm. per ton.

The effectiveness of antibiotics and sulfonamides against the psittacosis virus group, using the mouse pneumonitis virus (MPV), was studied.[17] Chlortetracycline, oxytetracycline (Terramycin†), chloram-

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*Aureomycin is produced by American Cyanamid Company, Pearl River, N. Y.

†Terramycin is produced by Chas. Pfizer and Co., Inc., Brooklyn, N.Y.

phenicol (Chloromycetin§), and erythromycin, when given orally, were highly effective in the prevention and treatment of MPV infections in mice. Penicillin G in large amounts suppressed the infection and eradicated the virus from the lungs. Sulfadiazine, sulfathiazole, and sulfamerazine were effective in the treatment of the pulmonary infections with sulfadiazine-sensitive strains. However, on serial passages in mice, MPV rapidly became resistant to sulfadiazine. Streptomycin has little activity against the psittacosis group of viruses.

CHEMOTHERAPY OF BACTERIAL DISEASES

Infectious Coryza.—This disease, caused by *Hemophilus gallinarum*, is characterized by an infection of the upper respiratory passages and conjunctiva. Fowl which recover without the benefit of treatment may become carriers. The most widely accepted treatment is sulfathiazole (0.5%) in an all-mash ration for five days or in the drinking water at 1:1,000 concentration for three to four days. Sodium sulfathiazole (0.27%), although it affected the palatability of the drinking water, did produce blood concentrations which were effective in the control of infectious coryza produced by inoculating chickens with *H. gallinarum*.[27] A 0.032 per cent solution failed to control the condition. Sulfamethazine in a 1:1,000 concentration in the drinking water has also been recommended. It is used for three days, omitted for three days, then used another three days. Sulfamethazine may also be used in the mash (0.4%) for five days.[15] Streptomycin given intramuscularly (0.2 Gm./bird) controlled coryza but smaller doses did not completely kill the pathogen and had a tendency to produce drug-fast strains even after one injection.[5,6]

Lugol's solution (1 dr./gal.) in the drinking water was reported to be a successful treatment.[18]

Fowl Cholera.—Pasteurella infection (*Pasteurella multocida*) has been a problem for many years. There is still no medicament which has been generally successful.

Sulfaquinoxaline has been used perhaps more frequently than any other drug. The recommendations for its use are: (1) Starve the birds for one half day. (2) Provide only drinking water containing sulfaquinoxaline (0.04%) for two to three days, or offer a feed containing sulfaquinoxaline (0.1%) for two to three days, or if feed intake has dropped, offer both a feed containing 0.05 per cent and water containing 0.025 per cent of sulfaquinoxaline. (3) If the disease recurs, use sulfaquinoxaline (0.025%) in drinking water or (0.05%) in the feed for two more days.

Sulfaquinoxaline (0.04 to 0.05%) in the drinking water or its continuous use in the feed (0.033%) has also been recommended.[15]

Sulfamerazine (0.2%) in the drinking water for five days, repeated after four days without treatment, has reduced the mortality in field cases. A 0.5 per cent concentration in an all-mash ration has also reduced high mortality.[15] Sulfamerazine (0.5%) in the mash promptly reduced the mortality in experimentally infected turkeys as well as in field cases, but the disease recurred and it was necessary to treat the flock on several additional occasions in order to control the condition.[1] Sulfamerazine, administered orally for two days at the rate of 143.0 and 107.25 mg. per kilogram of body weight, reduced the mortality in turkeys due to experimentally induced fowl cholera.[2] The disease reappeared after treatment was withdrawn. Also, sulfamerazine in the mash (0.4 or 0.3%) reduced the mortality in chickens artificially exposed to *P. multocida*.

Streptomycin was active against the infective agent isolated from turkeys.[19] These tests were made both *in vivo* and *in vitro*. The best results were obtained when 150 mg. (150,000 units) of streptomycin were given before or at the time of inoculation. Delay of treatment reduced the value of the drug. The death loss was reduced when 100,000 to 200,000 units of penicillin with 100 to 200 mg. of dihydrostreptomycin was given parenterally to visibly sick turkeys.[21] Chlortetracycline was shown to have activity against experimental infections.[16,24]

Pullorum Disease.—As a result of the pullorum-typhoid testing program, the incidence of this disease in many areas is small. In most cases of pullorum disease, it is recommended that the infected birds be killed rather than treated.

A number of reports have suggested that the sulfonamides will reduce mortality due to pullorum disease. The early use, in the

§Chlormycetin is produced by Parke, Davis & Co., Detroit, Mich.

drinking water, of sulfamerazine (0.2%) for five to seven days, or sulfamethazine in 1:1,000 solution for three to five days has been recommended.[15]

Streptomycin administered parenterally was reported to reduce the mortality and morbidity in chicks experimentally infected with *Salmonella pullorum*.[4] The bacteriostatic and bacteriocidal action of streptomycin on *S. pullorum* was studied *in vitro*.[7]

The value of the nitrofurans in the control of salmonellosis in turkeys and chickens has been the subject of many scientific papers. However, to use any of the available medicaments as a replacement for testing can only lead to trouble. The question of the masking of serologically positive reactions in pullorum-infected birds is still controversial. When furazolidone (NF-180*) (0.04%) was fed to carrier birds, 7 of 8 became negative to the agglutination test 60 to 84 days after the termination of the treatment.[12]

In the Delaware area, furazolidone is used at levels of 0.0055 to 0.011 per cent of an all-mash ration. This is equal to a 100- to 200-Gm. per ton level.

Fowl Typhoid.—This disease is caused by *Salmonella gallinarum*, a species akin to *S. pullorum*. The treatments are similar. The use of a sulfamethazine (15%) and sulfathiazole (85%) combination in the drinking water (1:1,000 concentration) has been suggested, but results were not outstanding.[15] It has been reported that favorable results were obtained by the use of 100 Gm. of furazolidone per ton of feed for a two-week period in typhoid of turkeys.[21] Furazolidone (0.02 to 0.04%), when fed continuously in the mash, was found superior to the sulfonamides, dihydrostreptomycin, chloramphenicol, oxytetracycline, and chlortetracycline in the treatment of experimental fowl typhoid.[20] There was a slight depression in the growth rate when fed at these levels for three weeks. Streptomycin, neomycin, magnamycin, chlortetracycline, oxytetracycline, tetracycline, and chloramphenicol were found to be active against *S. gallinarum*.[14] Chlortetracycline was more active than streptomycin *in vitro*, but 5 and 10 mg. of chlortetracycline given intraperitoneally was not effective in reducing losses in chicks experimentally injected with *S. gallinarum*.[20]

Sulfamerazine (0.4%) in mash or sodium sulfamerazine (0.2%) in the drinking water were effective in preventing losses produced by experimentally induced typhoid in chicks.[5] The treatment was given for seven days. However, the disease recurred on the third day after treatment was discontinued. When treatment was delayed until the infection was established, losses were severe during the next two days but stopped on the fifth day.

Furazolidone in the feed (0.011 and 0.0055%) was effective in preventing mortality associated with fowl typhoid infection in turkeys, especially at the higher level.[13]

The addition of 100 Gm. of this agent per ton of feed reduced mortality and controlled fowl typhoid in three flocks of turkeys and four flocks of chickens under field conditions.[5] Treatment at the level of 0.02 per cent in the ration prevented deaths of young chicks artificially injected with *S. pullorum* or *S. gallinarum*, but the diseases became evident when treatment was removed.[20] Better results were obtained with a 0.04 per cent level.

Erysipelas.—Although 100 and 200 mg. of chlortetracycline, given 24 and 48 hours after inoculation with *Erysipelothrix rhusiopathiae*, did not appreciably reduce mortality in turkeys, doses of 100,000 and 150,000 units of procaine penicillin were effective in eliminating mortality.[25] The results of *in vivo* and *in vitro* tests showed that penicillin, oxytetracycline, and a combination of penicillin and streptomycin were effective therapeutically.[20] Penicillin, however, showed the least variable effect on strains of the organism. Much of this work was done first in mice and later in turkeys. Penicillin gave the most consistent results. The use of 100,000 to 200,000 units of a long lasting penicillin by intramuscular injection was recommended for turkeys.[21]

Bluecomb Disease.—There is still considerable confusion regarding the nomenclature and cause of this disease. Almost all of the common medicaments have been recommended as a control or treatment for this condition in both chickens and turkeys. Until the cause is more clearly defined, there probably will be little clarity in the problem of treatment and control. Penicillin, streptomycin, oxytetracycline, and chlortetracycline in the drinking water were reported to be of value.[22] Following their use, mortality decreased 50 to 75 per cent from that of the untreated control

*NF180 is sold by Hess, Dr., & Clark, Inc., Ashland, Ohio.

groups. As a feed additive, 500 Gm. per ton of feed was necessary to produce beneficial results.

When a catarrhal enteritis of chicks was treated with the same four antibiotics at the level of 1 Gm. per gallon of drinking water, mortality was reduced 50 to 85 per cent.[23] Furazolidone at 0.0055 per cent was of no value. Bacitracin, dihydrostreptomycin, erythromycin, and other agents have also been recommended and used as treatments for bluecomb. In general, high levels are necessary for results. None of the antibiotics have been consistently beneficial in the treatment of bluecomb in Delaware flocks.

Chronic Respiratory Disease Complex.—The value of the many treatments and control measures now being recommended and used for chronic respiratory disease (CRD) is questionable. The results of controlled laboratory tests, as well as field-type experiments, have not indicated any outstanding therapeutic agent. Although a certain treatment may seem to produce beneficial results in one case, it may fail completely in another. This may be due in part to the complicated nature of the disease complex. It has been determined that the primary etiological agent is a pleuropneumonia-like organism (PPLO). However, in most field cases, a culture of the respiratory organs reveals the presence of one or more bacterial secondary infections and possibly a viral infection in addition to the PPLO.

Thus, the beneficial results seen in one case may be due to the ability of the medicament to control the secondary invaders, while its failure in another case may be due to its inability to control the secondary bacterial or viral infection, or both.

Infectious Synovitis.—It has been determined that 200 Gm. of chlortetracycline per ton of feed will control this disease,[10,22] but it is not beneficial for birds which are already crippled or lame.

Furazolidone at the level of 200 Gm. per ton has been recommended as a treatment. However, in a large number of field and laboratory experiments, chlortetracycline has proved most effective. This may be due in part to its ability to control a larger number of strains of the etiological agent. It has been shown that 100 Gm. of chlortetracycline per ton of feed is also effective prophylactically.[22]

That differences in drug susceptibility occur between strains has been shown experimentally and in natural field outbreaks.

Although oxytetracycline has not been tested by the author in as many experimental trials and field cases as chlortetracycline and furazolidone, it has proved to be an effective treatment.

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Treating Fowl Cholera in Turkeys.—When turkeys 12 weeks old were treated at the same time they were inoculated against fowl cholera, they were protected either by chloromycetin (1 Gm./lb. of feed or 0.2 Gm. intramuscularly), or with sulfaquinoxaline (1.5 Gm./10 lb. of feed or 1.5 oz. of solution per gal. of water). When treatment was delayed 18 hours after infection, only the intramuscular chloromycetin prevented losses, and when delayed until illness appeared (24 hr. after infection), two thirds of the birds thus treated recovered.—*Poult. Sci.* (*May, 1957*): *536.*

Possible Evidence of a Viral Agent in Shipping Fever Complex of Cattle

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Although neither complete nor conclusive, these observations may be of use to other workers in this field. While a viral agent has been suspected as the primary cause of shipping fever complex in cattle, there have been no reports of the recovery of such an agent.[1,2,3,4] Evidence for the possible existence of such an agent is presented.

Experimental Procedures and Results

Of the field cases of the disease studied, group A consisted of 30 experimental, yearling steers subjected to a change in feed. One developed a peracute respiratory infection and died, and 16 additional steers were then treated because of elevated temperatures and nasal discharge. Nasal washings (specimen A) were secured from 1 of these while it had a temperature of 105 F.

Group B consisted of 57 yearling Aberdeen Angus bulls shipped into the state six weeks previously. Three died suddenly, and when the fourth died it was presented for necropsy. Hemorrhages were present in the musculature of the cervical and costal areas and in the diaphragm. The anterior three fourths of the lungs was densely consolidated with a heavy fibrin on the pleural surface. There was an excess of straw-colored fluid in the pleural and pericardial cavities with fibrinous, pericardial adhesions. The mucosa of the abomasum and upper small intestine was congested; catarrhal enteritis was present in the lower part of the small intestine. *Pasteurella multocida* was recovered from the lung tissue. Pleural fluid (specimen B) was retained for experimental use.

Case C was an 8-year-old Holstein-Friesian cow with a temperature of 105 F. and signs of respiratory infection, although later the diagnosis was extended to traumatic gastritis. Fibrin shreds were evident in the nasal washings (specimen

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C) and *P. multocida* was recovered from this source.

The saline nasal washings (A and C) and pleural fluid (B), after treatment with penicillin and streptomycin, were inoculated into the yolk and allantoic sacs of chicken embryos that had been incubated for one week. After 48 hours' further incubation at 99 F., the allantoic fluid and yolk sacs were harvested. Most of the embryos were still alive. Employing 1 ml. of a 1:10 dilution of these embryo fluids from sources A and B and 1 ml. of 2 per cent washed sheep cells, some settling occurred after one hour at room temperature, but definite hemagglutination, based on the pattern of cells with only minor tilting, could be discerned with the allantoic fluids after 24 hours at room temperature.

Subsequently, second passages of allantoic fluid were made from sources A, B, and C into the allantoic sac of developing chicken embryos. The harvested allantoic fluids were set up in twofold dilutions progressing from 1 to 8. Then 0.25 ml. of saline and 0.5 ml. of either 1 per cent washed chicken cells or 1 per cent washed lamb cells were added to 0.25 ml. of each dilution. Normal allantoic fluid from uninoculated embryos and the allantoic fluids from the second passages of A, B, and C all showed normal settling of the red cells of both species at one hour and overnight at room temperature. At the same time, first passages of B and C were included. C was negative but B produced hemagglutination of chicken cells, to a dilution of 1:16 at one hour and 1:32 overnight and of lamb cells to 1:32 at both reading times. This same procedure, using adult sheep cells and the first allantoic passage of C and second of A and B, also produced negative results. Inhibition of this hemagglutination of lamb cells was demonstrated by "convalescent" serum from the steer supplying material for A, but neither by the "acute" serum from bull B nor by two randomly selected serum samples submitted to the laboratory for brucellosis testing.

CONCLUSIONS

Nasal washings or pleural fluid from a yearling in each of two groups of cattle which developed a highly contagious acute respiratory infection, following shipment or change of feed, when treated with antibiotics and inoculated into the allantoic sac of embryonating eggs, yielded material which hemagglutinated chicken and sheep cells. This property did not carry over into a second passage through allantoic sacs. This phenomenon would seem unrelated to the false positive secured with allantoic fluids inoculated with unfiltered throat washings.[3] These false positives associated with bacterial contamination of the inoculum are of a labile nature and did not survive four days at 4 C., whereas the agents used were stored for variable periods beyond this without loss of hemagglutination. Normal human and rabbit serums also inhibit the formation of such false positives. While serums from these species were not employed, normal cattle serums did not inhibit the observed hemagglutination.

While this study is necessarily incomplete, it would suggest that a viral agent was recovered by inoculation of embryonating eggs but could not be maintained there. Its presence was demonstrated by hemagglutination of sheep and chicken cells.

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Parasitic Gastritis in Housed Cattle.—Severe chronic diarrhea and emaciation in heifers 12 to 18 months old, on ten farms in Scotland, was found to be caused by heavy abomasal infection with nematodes, almost entirely *Ostertagia ostertagi*. The condition started in the winter and continued into the early summer. On eight farms, the animals were kept in stalls throughout the winter. In addition to a watery diarrhea, with no evidence of hemorrhage or tenesmus, there was loss of weight, anorexia and, in some cases, a subcutaneous submaxillary edema. In some cases, there was mild anemia and the erythrocyte count and hematocrit values were low. In the fundic and pyloric areas of the abomasum, the mucosa was thick-

ened and granular and the submucosa was often grossly edematous. The associated lymph nodes were enlarged.

The clinical signs usually developed in three to ten weeks, but occasionally five months, after the animals were brought in from pasture. In the cattle with fatal cases, many of the parasites were immature. Their development may have been inhibited by resistance acquired from infection when the cattle were placed on pasture the previous summer.—*W. B. Martin, et al. Vet. Rec. (Aug. 3, 1957): 736.*

Preventing Traumatic Gastritis with Bar Magnets.—Bar magnets were given orally to 34 cows. In 12 of the 17 which had not been deprived of hay, the magnets were found, upon rumenotomy 30 minutes later, in the anterodorsal sac of the rumen of 11, and in the reticulum of 1; in the other 5, all of the magnets were in the anterodorsal sac of the rumen after 24 hours.

In the 17 cows deprived of hay for 24 hours prior to administration, 24 hours after administration the magnets were recovered from the reticulum of 10 cows and from the anterodorsal sac of the rumen of 2; after 48 hours, the magnets in the other 5 cows were recovered from the reticulum.

In 4 other cows, given magnets while receiving hay and then given a capsule full of sharp nails and wire 30 minutes to ten days later, rumenotomy, 15 to 120 days later, revealed the metal attached to the magnet in all 4—1 was in the anterodorsal sac, 1 in the posterior compartment of the rumen, and 2 in the reticulum.

The final 4 cows were deprived of hay for 24 hours then given a magnet and, 30 minutes to seven days later, a capsule full of metal. All magnets with the metal attached were found in the reticulum 41 to 150 days later.

It was concluded that the animals should be deprived of hay for at least 24 hours before magnets are given.—*R. L. Lundvall in a report from the Veterinary Medical Research Institute, Iowa State College (1956): 82.*

Toxicity of Organic Phosphorus Insecticides.—Recently produced organic phosphorus insecticides have a lower toxicity for animals than earlier products. A case is recorded, in Britain, where a number of steers showed a reaction within 15 to 30 minutes after being sprayed with a potent thiophosphate for tick control. They showed staggering, stiffness, pushing with their heads, protruding tongues, profuse salivation, labored respiration, and blindness due to constriction of the pupils. Some collapsed and died within 30 minutes without struggling. Others were recumbent for five to six hours, then recovered. Poisoning of sheep, pigs, dogs, and poultry is also described. Poultry are said to be more susceptible than mammals. The recommended treatment is double the therapeutic dose of atropine, intravenously. Sprayed animals should be washed. Saline purgatives may be indicated, but not oils. In carnivores, emetics are indicated if vomiting has not occurred.—*D. W. Jolly in Vet. Rec. (Aug. 24, 1957): 796.*

Hygromycin, a Broad-Spectrum Anthelmintic for Swine.—In a helminth count made on 101 swine marketed in Wisconsin, in a recent two-year period, 46,797 parasites from 11 genera were found. The average hog carried 463 helminths representing 3.5 genera. A greater variety of parasites was recovered from the smaller hogs; a greater number (av. 208 more) from the heavier hogs. This indicates that helminth control measures should include breeding stock.

In an experiment with 5 pigs, 4 of them stunted and all of them heavily parasitized, the antibiotic, hygromycin, was found to be an effective anthelmintic against the lungworms, whipworms, and strongyloides and, possibly, the ascarids. Ova production by all of these species, also by the stomach worms, showed marked reduction after about 14 days. These pigs were given 2 Gm. of purified hygromycin per 100 lb. of ground feed for 54 to 72 days before they were slaughtered, the total of hygromycin per pig being from 2.8 to 4.8 Gm. One of the pigs died from parasitism before the drug could be effective. Another was so weak, emaciated, and anoretic that it was given 0.06 Gm. of the drug *per os* daily for six days. Its appetite returned in a few days and it began to gain weight. During the period of medication, the weight gain of the 4 pigs averaged 1.1 lb. per day. They showed no signs of intoxication.—*A. I. Goldsby and A. C. Todd, University of Wisconsin.*

Experimental Oral Transmission of Vibrionic Abortion of Sheep

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A great deal of research has been conducted in an attempt to determine a mode of transmission of vibrionic abortion which might account for the high percentage of abortions experienced in sheep under natural conditions. The vaginal route has been explored extensively with essentially negative results. This is in distinct contrast to the disease produced by *Vibrio fetus* in cattle which appears to be primarily a venereal infection. This paper reports on the successful experimental oral transmission of the disease to a high percentage of susceptible ewes.

The oral administration of *V. fetus*-infected ovine uterine exudates and fetal material to 26 ewes resulted in no abortions, but vibrionic infection of the uterus was established in 4 ewes as shown by necropsies of the ewes slaughtered at varying intervals afterward.[5] Two of 3 ewes drenched with *V. fetus*-infected fetal fluid and vaginal discharge aborted, and *V. fetus* was recovered.[3] Also, the oral administration of infected placental tissue and infected fetal stomach contents to pregnant ewes at the Montana Veterinary Research Laboratory resulted in infection.[9]

In the spring of 1955, 17 of 23 ewes fed infected tissue during the early part of the fifth month of pregnancy aborted, and *V. fetus* was recovered from all of the aborted fetuses.[2]

During the same year, when ewes were inoculated orally with *V. fetus* cultures, at different stages of gestation, 8 of the 12 ewes inoculated at 92 to 97 days of pregnancy aborted and 7 of the 12 ewes inoculated at 124 to 127 days aborted.[7]

Other routes of transmission of the disease have been studied extensively with essentially negative results, although some evidence has been presented that intravaginal inoculation may result in infection. At Colorado State University,[1] one abortion occurred in a group of clean ewes bred to infected rams and *V. fetus* was isolated from the aborted fetus. At the Montana Veterinary Research Laboratory,[8] uterine infection, but not abortion, resulted in 2 of 27 ewes inoculated intravaginally at the time of breeding. One ewe died of unknown cause three days after inoculation and *V. fetus* was isolated from the uterus. *V. fetus* was isolated from uterine swabs made after parturition in 1 of 21 of the above ewes which lambed normally.

Materials and Methods

On Feb. 26, 1956, 24 range-bred yearling ewes, with no known exposure to *V. fetus*, were purchased from a range band into which rams had been turned on Dec. 15, 1955. The ewes were allotted to two groups of 12 each. Group I served as noninfected controls. Each ewe in group II was inoculated orally on March 11, 1956, with 1½ oz. of a suspension prepared from placentas, stomach contents, and livers of 2 aborted fetal lambs from which *V. fetus*, of type I strain, had been isolated.* These fetuses were obtained from a band of 900 ewes, 15 per cent of which had aborted.

The two groups of ewes were maintained in separate pens with a 12-ft. alley between and with separate water and feeding facilities. The ration fed both groups was 5 lb. of alfalfa hay plus ½ lb. of a cubed 20 per cent protein supplement per ewe daily.

Results

In group I, 1 of the 12 ewes (8%) aborted twin lambs. Both lambs were culturally negative for *V. fetus*.

In group II, 11 ewes (100%) aborted (1 ewe apparently failed to conceive). The average incubation period was 19 days (table 1). *Vibrio fetus* was isolated from 13 of the 15 aborted fetal lambs from this group.

TABLE I—Days from Oral Inoculation with V. Fetus to Abortion

Days from inoculation	9	10	12	13	16	18	28	30	53
Number of abortions	1	2	1	1	2	1	1	1	1

Discussion

The ewes which had been bred the day the rams were turned into the range band would have been 87 days pregnant on the date group II was inoculated. In as much as the ewes in group I completed lambing in approximately 15 days, it can be inferred that the stage of gestation in group II at

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*Typed by B. D. Firebammer of the Montana Veterinary Research Laboratory. The type I strain is most commonly isolated, by the Montana workers, from ovine aborted material.

the time of inoculation was 72 to 87 days. This agrees with the findings of others[2,7] that susceptible ewes inoculated orally with *V. fetus* cultures or *V. fetus*-infected tissues during the third, fourth, or fifth month of gestation may abort.

SUMMARY

Of 12 yearling ewes inoculated orally with *Vibrio fetus*-infected tissues during the last half of the third month of gestation, 11 aborted and the twelfth was nonpregnant. The average incubation period was 19 days. *Vibrio fetus* was isolated from 13 of the 15 fetal lambs aborted by the infected ewes. Of 12 control ewes, only 1 aborted and her twin lambs were culturally negative for *V. fetus*.

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Sheep Passage of Equine Infectious Anemia.—Of 4 sheep inoculated with blood from a foal with equine infectious anemia (swamp fever), 3 developed a syndrome resembling the disease in horses. In the second sheep passage, 2 of 4, and in the third passage, none of 4, were affected. None of 4 foals injected with blood from 4 of the affected sheep showed clinical reactions but all were resistant when challenged with a weakly infective virus.—*Arch. Exptl. Vet.-med., 10, (1956): 709 (abstr. in Vet. Bull. (Aug., 1957): Item 2381).*

Control of Psittacosis in Parakeets.—Incipient psittacosis can not be detected in psittacine birds except by virus isolation, which requires killing the bird. Healthy appearing parakeets may spread the virus throughout their lives. This latency is essential for the psittacosis parasite to maintain its infection chain. Some young infected birds become free of the virus within a few months, others never do and may become shedders when subjected to certain stresses such as different environment, excessive fondling, or chilling.

In one instance, a parakeet fatally infected four persons before its death from psittacosis. Its apparently healthy cagemate was then placed in a glass cage and a succession of highly susceptible ricebirds were introduced at intervals. Those introduced during the first six months died of psittacosis. After ten months, this parakeet was killed and the virus was recovered from the spleen and liver at necropsy. All parakeets offered for sale should be treated by feeding a tetracycline compound—not less than 30 mg. in 15 days.—*J.Am.M.A. (Sept. 7, 1957): 113.*

Infectious Bronchitis and Chronic Respiratory Disease.—Birds in a laying flock, brooded and raised on an isolated island, were vaccinated with a combined Newcastle disease-infectious bronchitis vaccine at 8 days of age and antibody levels for both viruses were determined at intervals for a year. Substantial levels of antibody were maintained. The flock was then exposed to infectious bronchitis virus and the resulting inapparent infection reactivated chronic respiratory disease which was latent in the flock.—*Vet. Bull. (Aug., 1957): Item 2406.*

Effect of Nicarbazin on Eggs.—The bleaching action of nicarbazin (a coccidiostat) on brown-shelled eggs might suggest its use to improve the color of eggs for the market. When fed at a level of 0.0125 per cent of the ration, the eggs from some birds were almost completely white after three days but egg production later was reduced about 8 per cent and egg weight about 5 per cent. After ten to 15 days, an objectionable yolk mottling occurred in some eggs.—*Poult. Sci. (May, 1957): 589.*

Further Experiences with Furacin in Treatment of Ovine and Caprine Coccidiosis

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In a previous report,[5] we described the results obtained with nitrofurazone N.F. (Furacin*) in the treatment of lambs and kids affected with coccidiosis due to *Eimeria faurei*, and found that the powdered crystalline form of it, given orally, 7 and 10 mg. per kilogram of body weight daily for 7 days, was highly effective.

In the first experiment, 1 tablet daily for five days was only moderately effective in kids weighing an average of 5 kg. (11 lb.). Mortality was prevented but diarrhea often continued in reduced form. In later trials with 2 tablets per animal per day, diarrhea ceased during treatment and did not recur.

SUMMARY AND CONCLUSIONS

Nitrofurazone is highly effective in the treatment of ovine and caprine coccidiosis caused by *Eimeria faurei*. Furadex tablets are more convenient for this purpose than the powdered drug. Two tablets (100 mg.) daily for five days effectively prevented mortality during epizootics of *E. faurei* involving over 500 lambs and kids.

TABLE I—The Effect of Nitrofurazone Treatment in Coccidiosis of Goats and Sheep

	Animal treated		Daily dosage		Duration (days)	
District	Kind	No.	Tablets per animal	Nitrofurazone (mg./kg.)	Treatment	Diarrhea
Island of Oebea	Kids	100	1	10	5	4
Island of Oebea	Kids	150	2	20	5	3
Attica	Lambs	65	2	20	5	3
Phthiotis	Kids	170	2	20	7	3
Phthiotis	Kids	60	2	20	7	3

Later, working with the same disease in the Belgian Congo,[3] similar results were obtained. The drug has been reported effective in several other types of mammalian intestinal coccidiosis, such as bovine (*E. zurnii*),[4] porcine (*E. debliecki*),[2] and canine coccidiosis.[1]

Because of difficulties in using the powder form, tablets containing 50 mg. of nitrofurazone in an excipient of dextrose (Furadex tablets*) were used. Clinical trials were carried out on the Island of Oebea and the Districts of Attica and Phthiotis of Greece, during the lambing and kidding seasons of 1956 and 1957, by administering 1 or 2 tablets per animal daily for five or seven days. Studies revealed that *E. faurei* was the causal agent in all instances.

The evaluation of treatment was based solely on clinical criteria, *i.e.* cessation of diarrhea, reestablishment of the appetite, and improvement of the general condition (table 1).

The condition of the diseased animals improved on the third day and recovery was complete on the fifth day of treatment.

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The authors thank Drs. H. D. B. Roberts and P. Veloglu for their assistance and cooperation.

*Furacin, a brand of nitrofurazone N.F., and Furadex tablets were furnished by Eaton Laboratories, Norwich, N.Y.

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Cattle Production Cycles.—The number of cattle in the United States in 1957 is estimated to be about 1.6 per cent less than the cyclical peak of 96.8 million in 1956, which was 20 million (26%) more cattle than in 1949. The downtrend is expected to end about 1960 at around 92 million head, a considerably smaller decrease than in the cycles of recent decades. The downtrend which started in 1918

(22%) continued ten years; those starting in 1934 (12%) and in 1945 (10%) each lasted four years. The average beef consumption per person hit a low point in 1951 (56.1 lb.) and reached a peak in 1956 (85.4 lb.) but is not expected to drop below 76 lb. in 1960. In 1956, central markets handled 59 per cent of marketing cattle sold, 71 per cent of those slaughtered, and 98 per cent of all federally inspected beef cattle.—*Am. Cattle Producer (Sept., 1957): 9, 24.*

A Comparison of Tests for Bovine Brucellosis.—Thirteen cows known to be infected with *Brucella abortus* were tested, by three methods, every two weeks for six months, a total of 261 tests with each method. Of the 261 complement-fixation tests, 200 were positive, 56 negative, and 5 doubtful; of the agglutination tests, 127 were positive, 95 negative, and 39 doubtful; with the ring test, 162 were positive, 98 negative, and 1 doubtful. The ring test was favored because it was simple, easy to interpret, and there was no drop in milk yield due to handling the animals.—*Vet. Bull. (Aug., 1957): Item 2322.*

Subcutaneous Brucellosis Vaccination in the Ear.—Cattle were experimentally vaccinated with strain 19 vaccine, in South Africa, by an injection into the subcutaneous tissue of the ear. The vaccine was reconstituted so that 1 ml. contained a full dose. The vigorous ear movements after injection were believed to assist in spreading the vaccine and minimizing local reaction. Without interfering with the immunizing effect, this method seemed to decrease: (1) the local inflammatory reaction; (2) functional disturbances; (3) risk of permanent blemish; and (4) risk of abortion in pregnant cows. Also, it is convenient and requires no special syringe. Possible disadvantages are: (1) leakage of the vaccine from the thin skin; and (2) the care required to prevent injection into the conchal cartilage.—*J. South African V.M.A. (June, 1957): 119.*

Exercise Myohemoglobinuria.—Five male students, 20 or 21 years of age, at a midwestern university, developed painful, swollen muscles, and a coffee-colored urine about 24 to 48 hours after strenuous exercise (knee-bends, push-ups, etc.) to the point of exhaustion, as a result of fraternity hazing or friendly competition. All showed albumin and varying numbers of leukocytes in the urine, and one had a mild fever and a few erythrocytes in the urine.

The mechanism of hemoglobinuria is still unknown but there is probably blood destruction intravascularly and a breakdown of muscle fibers due to extreme exercise. A total of 75 cases of "march hemoglobinuria" have been reported since 1941 (many in the German army) but, in these, hemoglobin appeared in the urine one to three hours after exercise. No cases have been reported among women or in other than the white race.—*J.Am.M.A. (July 27, 1957): 1458 (see* JOURNAL, *March 15, 1957: 237).*

Bracken-Poisoning Treatment. — Three experimental cases and many field cases of bracken poisoning in cattle were successfully treated with DL-batyl alcohol. This was given as four or five daily intravenous injections of 25 to 50 ml. of 2 Gm. of batyl alcohol with 5 Gm. of "tween 20" in 100 ml. of 1 per cent saline solution, or as subcutaneous daily doses of 1 Gm. of batyl alcohol in 10 ml. of olive oil.—*Biochem. J., 65, p. 5P of Proceedings; abstr. in Vet. Bull. (July, 1957): Item 2209.*

Swine-Testing Programs. — Testing of swine, to determine the strain which will produce the best lean carcass most economically, is spreading from the corn belt to other states. The first testing station was set up by a local organization in Illinois in 1954. This year, in Illinois, 225 litters are being tested in ten stations. By 1955, similar testing stations were set up at Ohio State University and near Iowa State College. One is now operating at Clemson Agricultural College in South Carolina. Several other states are planning similar stations.—*Nat. Hog Farmer (Sept., 1957): 6.*

Strongyloidiasis Fatal in Man.—A fatal subacute obstruction of the duodenum of a man, in Brazil, was believed caused by the local invasion of the mucosa and submucosa by the female parasites and larvae. Vascular or possibly nervous lesions could have been responsible for the spasm in the gut. —*J.Am.M.A. (July 27, 1957): 1498.*

Observations on the Chemotherapy of Canine Heartworm Infection with Arsenicals

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AS A RESULT of extensive work published in more than 50 papers in Japanese and summarized recently, in the *American Journal of Veterinary Research*,[9] it has been concluded that trivalent arsenic in the form of dichlorophenarsine hydrochloride (Filarsen) is superior to other chemicals tested so far for the control of dirofilariasis. The comparative activities of this compound, trivalent antimonials, and 1-diethyl carbamyl-4-methyl piperazine (Supatonin) are shown (table 1). The adult worms were destroyed by dichlorophenarsine hydrochloride in three daily doses of 1.0 mg. As per kilogram of body weight.

TABLE I—Comparison of the Activities of Three Therapeutic Agents Against Three Stages of Dirofilaria Immitis

Agents	Microfilaria	Developing stages	Adults
Antimonials	+++	+	+
Diethylcarbamazine	++	± ?	± ?
Dichlorophenarsine	−	+++ ?	+++

The destruction of the adult worms in 15 days by thiacetarsamide (arsenamide, Caparsolate sodium) has been reported[11-14] using the same total dose (3.0 mg. As/kg.), but administered as 15 daily doses of 0.2 mg. As/kg. Failure to kill the adult worms within five days with the same total administered over a three-day period was also reported.[13]

We have found that with dichlorophenarsine,[6-10] or the antimonials,[2,7,8,10] some minimum period of time is required to obtain the full effect of the drug used upon the adult worms and, when the drugs are administered in a single dose or a few daily doses, that this period usually extends beyond the time of actual treatment. It may be months after treatment before the antimonials kill the adult worms but, even with dichlorophenarsine, ten days or more may be required. We assumed, therefore, that thiacetarsamide, which like dichlorophenarsine is a substituted phenyl arsenoxide, might require a comparable period to destroy the adult worms regardless of the length of time over which the drug is administered. A total of 3.0 mg. As/kg. in the form of thiacetarsamide might require up to 15 days to kill the worms, but the treatment need not necessarily be divided into 15 daily doses.

The object of this paper is to present a comparison of the therapeutic actions and the toxicities of dichlorophenarsine and thiacetarsamide.

MATERIALS AND METHODS

Both arsenicals were given intravenously in doses of 1.0 mg. of As/kg. daily unless otherwise indicated. The microfilariae were counted in 0.01 cc. of blood before, during, and after treatment. The fate of the adult worms was determined at necropsy. Since, in our previous work, only dead worms were found in dogs killed ten or more days after initiation of treatment with three daily doses of 1.0 mg. of As/kg., none of the animals in these studies was killed until at least ten days had elapsed since the beginning of treatment.

RESULTS

Dichlorophenarsine Therapy.—The results of these experiments (table 2), and those reported previously[9] on dogs which received one or more doses of 1.0 mg. of As/kg., are summarized (table 3). It will be seen from these tables that all the adult worms were dead when five or more doses of 1.0 mg. of As/kg. had been administered, even when as little as five days elapsed between the first dose and necropsy. Over 94 per cent of the worms were dead in the dogs that were given three such daily injections and were held at least ten days before necropsy. In our previous studies, we found only 4 living and no dead worms in one such dog that was held only five days after treatment.[9] In the present series of experiments, 2 dogs were given

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three doses of 1.0 mg. of As/kg. on alternate days and were held 13 and 15 days. The former (dog 52, table 2) harbored 60 dead and no living worms while the latter (dog 51) had 1 living and 3 dead worms. When only two daily doses were given, all the worms were dead in the 3 dogs held 20 to 30 days, and 3 of the 5 were dead in the dog which was killed 15 days after the first treatment.[9] After a single dose of 1.0 mg. of As/kg., only living worms were recovered even though the dogs were held 25 and 30 days.

In our previous studies,[9] it was found that a dog given a single dose three times this size and held ten days yielded only dead worms, but a dog which died two days after treatment yielded 4 living worms and 1 dead worm. With daily doses of less than 1.0 mg. of As/kg., the results were more erratic and less promising (table 2). Of the 2 dogs (table 2) given as little as three daily doses of 0.75 mg. of As/kg., 1 (dog 42) harbored only dead worms at necropsy 20 days later, but 2 living and 2 dead worms were found in the other (dog 43).

The number of microfilariae remained essentially unchanged before and after treatment (table 2).

Toxicity of Dichlorophenarsine.—Daily doses of 1.0 mg. of As/kg. were well tolerated by all except dog 21 (table 2). Even the dog in our previous studies, which was given 15 such daily doses, suffered no serious side effects. In some cases, signs of nausea, vomiting, or transient excitement occurred immediately after injection, but these reactions subsided quickly and were never severe enough to be considered as contraindications to continued treatment. One of the 9 dogs (dog 21, table 2) given five daily doses of 1.0 mg. of As/kg. died five days after the first dose. One of the 2 dogs, in our previous studies, given a

TABLE 2—Treatment of Dirofilariasis with Daily Injections of Dichlorophenarsine Hydrochloride

Dog No.	Daily dose (mg. As/kg.) and No. of injections	Total dose (mg. As/kg.)	No. of microfilariae in 0.01 cc. blood		Days between first injection and necropsy	Adult worms	
			Before treatment	After treatment		Alive in heart	Dead in pulmonary artery
16	1.0 x 5	5	—	—	40	—	Many
46	1.0 x 5	5	203	—	38	—	Many
47	1.0 x 5	5	11	—	38	—	Many
17	1.0 x 5	5	—	—	27	—	Many
18	1.0 x 5	5	29	29	25	—	Many
19	1.0 x 5	5	—	—	22	—	20
48	1.0 x 5	5	29	29	22	—	Many
20	1.0 x 5	5	—	—	20	—	Several
21	1.0 x 5	5	240	—	5D	—	70
22	1.0 x 3	3	309	—	30	2	15
23	1.0 x 3	3	222	—	21	—	38
49	1.0 x 3	3	—	—	17	—	3
24	1.0 x 3	3	32	9	15	—	Many
25	1.0 x 3	3	171	252	15	2	12
26	1.0 x 3	3	47	51	10	—	Many
27	1.0 x 3	3	140	—	10	—	9
28	1.0 x 3	3	37	26	10	—	5
29	1.0 x 3	3	—	—	10	—	9
30	1.0 x 3	3	—	—	10	—	5
31	1.0 x 3	3	—	—	10	—	13
32	1.0 x 3	3	270	551	10	—	38
33	1.0 x 3	3	4	13	10	—	3
34	1.0 x 3	3	1	1	10	—	4
35	1.0 x 3	3	16	70	10	—	39
36	1.0 x 3	3	25	56	10	3	6
37	1.0 x 3	3	21	44	10	4	4
38	1.0 x 3	3	9	2	10	1	9
39	1.0 x 3	3	—	—	10	1	6
51D	1.0 x 3	3	1	4	15	1	3
52D	1.0 x 3	3	114	486	13	—	60
40	1.0 x 1	1.00	124	123	25	11	—
42	0.75x 3	2.25	49	24	20		10
43			6	—	20	2	2
45	0.75x 2	1.50	84	—	30	1	15
50	0.65x 5	3.00	220	146	10	3	8
44	0.5 x 4	2.00	139	665	20	8	More than 23
41	0.3 x 10	3.00	33	42	32	8	8

DTreated every other day; D=died.

TABLE 3—Results of Treatment with Dichlorophenarsine Hydrochloride

mg./kg. x No. doses	Days to necropsy	Dog (No.)	Freed of infection (%)	No. of worms		Dead worms (%)
				Alive in heart	Dead in pulmonary artery	
1 x 7-15	7-15	11	100	0	>104	100
1 x 3	10-30	22	73	15	>234	>94
1 x 3	5	1	0	4	0	0
1 x 2	20-30	3	100	0	Many	100
1 x 2	15	1	0	2	3	60
1 x 1	25-30	2	0	12	0	0

Taken from table 2 of this manuscript and from Table 9 in "Chemotherapy of Canine Filariasis," by S. Kume, in Am. J. Vet. Res., 18, (Oct., 1957): 912-923.

single dose of 3.0 mg. of As/kg. died two days later. The reactions were apparently due to hepatic dysfunction.

Thiacetarsamide Therapy.—At necropsy of 10 of the dogs given three daily doses of 1.0 mg. of As/kg. and killed ten days later, 97 per cent of the worms were dead. No living worms were found in 8 of these 10 dogs (table 4). The 1 animal (dog 11) which died five days after the first injection was the only one harboring living but no dead worms. Only dead worms were found in 2 dogs (12 and 13) which were given only two such doses but were held for 20 days. Lower doses were not tried.

Again, as following treatment with dichlorophenarsine, there was no evidence of any change in the number of microfilariae.

Toxicity of Thiacetarsamide.—The vomiting and transient excitement, which occasionally occurred immediately after injection, was no more frequent or severe than those following the injection of dichlorophenarsine. However, 3 dogs (3, 6, and 7) developed icterus, and marked hepatic fatty degeneration was found at necropsy in another (dog 11) which had died. Such evidence of severe liver damage was not seen following the same dosage of dichlorophenarsine.

DISCUSSION

Dichlorophenarsine and thiacetarsamide, with identical doses of arsenic, appear to have essentially the same effect upon the adults of *Dirofilaria immitis*. Over 94 per cent of the worms were dead when recovered from dogs at necropsy, ten days or more after beginning treatment with dichlorophenarsine at the rate of 1.0 mg. of As/kg. daily for three days. It was reported[13] that thiacetarsamide, in daily doses of 0.2 mg. of As/kg. for 15 days, killed essentially all the heartworms, but others[1] found that twice this daily dose for the same period was required to kill all the worms. It appeared probable to us that much the same result could be accomplished by compressing this dose schedule into three days, just as with dichlorophenarsine. Ten days after the beginning of treatment with thiacetarsamide, at the rate of 1.0 mg. of As/kg. daily for three days, 97 per cent of the worms recovered were dead. The method differed from that used in 1948[13] in that, following the compressed period of treatment, the dogs, in 1948, were necropsied three and five days after the initiation of therapy.

An inverse ratio between dosage and the

TABLE 4—Treatment of Dirofilaria Immitis with Daily Injections of Thiacetarsamide

Dog (No.)	Daily dose (mg. As/kg.) and No. of injections	Total dose (mg. As/kg.)	No. of microfilariae in 0.01 cc. blood		Days from first injection to necropsy	Adult worms	
			Before treatment	After treatment		Alive in heart	Dead in pulmonary artery
1	1 x 3	3	61	82	10K	----	10
2	1 x 3	3	4	4	10K	----	13
3	1 x 3	3	24	21	10IK	----	5
4	1 x 3	3	9	5	10K	----	2
5	1 x 3	3	---	128	10K	----	9
6	1 x 3	3	241	200	10IK	----	10
7	1 x 3	3	947	1005	10IK	----	46
8	1 x 3	3	491	554	10K	----	19
9	1 x 3	3	53	30	10K	2	10
10	1 x 3	3	15	34	10K	2	9
11	1 x 3	3	197	128	5D	24	---
12	1 x 2	2	174	277	20K	----	22
13	1 x 2	2	41	55	20K	----	12

K = healthy killed; IK = icterus developed, killed; D = died.

time required to kill the worms had been demonstrated previously for the trivalent antimonials.[9] It is impractical to use these compounds for the destruction of the adult worms, but a similar principle seems to hold for the arsenicals. A single dose of 3.0 mg. of As/kg. killed all the worms in ten days and the same total dose, given as three daily 1.0-mg. doses, killed nearly all of them in the same period; yet only 1 out of 5 worms was killed in two days by a single 3.0-mg. dose and none was killed in five days by three daily 1.0-mg. doses. A total of 5.0 mg. of As/kg., given as five daily 1.0-mg. doses, killed all worms within five days. As few as two daily doses of 1.0 mg. of As/kg., either as dichlorophenarsine or as thiacetarsamide, killed all the worms in 20 days, although at 15 days 2 of 5 worms were still alive in 1 dog. Three doses of dichlorophenarsine, as low as 0.75 mg. of As/kg., killed most of the worms in 20 days. This dose is considered more suitable for routine clinical use and is preferred by Japanese veterinarians.

It is important to carefully consider the state of the dog's health before treatment. The vomiting or transient excitement that sometimes occurs immediately after injection is of no great concern. However, if the dog is already suffering from pathological changes in the liver, or is harboring an extremely large number of heartworms, it is a poor therapeutic risk. The fewer mosquito seasons the dog has gone through, the less the risk attending therapy. Over several seasons, there is danger of liver changes from accumulated worms; also an increasing chance that the dog is harboring large numbers of worms which could produce pulmonary emboli after successful treatment.

From the standpoint of the therapeutic response, the disease may be classified in three stages:

First Stage.—The first stage is one of early infection with comparatively few worms of less than maximum size. Treatment should be both safe and successful since the danger of liver damage is nil and the danger from post-treatment pulmonary embolism slight.

Second Stage.—This stage is one of longer standing infection but comparatively few worms. Treatment should still be successful; there is increasing danger of hepatitis but post-treatment pulmonary embolism should not be serious.

Third Stage.—The third stage is that of long-standing heavy infection. Treatment may constitute a real danger both from post-treatment hepatitis and pulmonary embolisms.

We consider it advisable, in Japan, to treat dogs annually in January. We have found that 1 to 21 worms (av. 4) develop from one summer's exposure in Japan and will have reached the heart by January. The worms are more readily destroyed by therapy after reaching the heart than during their intermediate developmental period. Yet, in January, they will not have reached their maximum size so that the danger from post-treatment pulmonary embolism is at a minimum. Furthermore, the infection will be of short duration so that the chances of injury to the liver or other internal organs will be slight.

SUMMARY

More than 94 per cent of the adult worms are destroyed within ten days by dichlorophenarsine hydrochloride (Filarsen) given intravenously at the rate of 1.0 mg. of arsenic per kilogram of body weight daily for three days. Thiacetarsamide (Caparsolate sodium) was found to accomplish the same result (97% destruction of the adult worms) when given at the same dosage and in the same manner. Thus, the more protracted treatment schedule of 0.2 to 0.4 mg. of As/kg. daily for 15 days is not required. A minimum of ten days must elapse before the full value of the arsenical can be determined.

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Penicillinase for Penicillin Reactions.—Penicillinase, an enzyme that destroys penicillin *in vivo* as well as *in vitro*, was given intramuscularly to 42 patients who had reacted unfavorably to penicillin. The effect was difficult to evaluate but some cases were dramatically relieved within a few hours.—*J.Am.M.A. (Sept. 21, 1957): 222.*

Atrophic Rhinitis Carried by Rats.—Pigs contracted atrophic rhinitis when inoculated with material from rats that had been previously infected with material from affected pigs. They developed the disease quicker (15 to 45 days) after being inoculated with material from rats than with material directly from affected pigs (24 to 51 days). Two thirds of all pigs experimentally exposed developed the disease.—*U.S.D.A. Release, Aug. 28, 1957.*

Re-establishment of Intestinal Flora.—A physician in Austria reports good results from using a preparation containing an emulsion of living *Escherichia coli* in 89 children after the normal intestinal flora had been destroyed by antibiotic therapy or after acute or chronic enteritis. —*J.Am.M.A. (Aug. 24, 1957): 1951.*

Cattle Identified by Blood Type.—Analysis of cattle blood types can be used to safeguard the registration system for purebred cattle. As in human blood, the typing of cattle blood depends on its anti-antibodies when blood from one animal is introduced into another animal. Cattle have about 50 antigenic factors, which can be arranged into millions of combinations. All bulls used for artificial insemination in the United States are typed at the School of Veterinary Medicine, Davis, Calif. The antigenic factors derive their identity from specific genes and, although there are hundreds of genes, only a few can be present in 1 animal. It is improbable that 2 animals, other than identical twins, would have the same blood type. By reference to the blood type of the dam and sire, the parentage of a calf may be determined.—*U.S.D.A. Release, Aug. 28, 1957.*

Swine Influenza in Ohio.—Field reports during 1956 indicated that swine influenza occurred on 910 premises in Ohio, an average of 76 per month. Surprisingly, over one fourth of these were reported during the spring and summer months, an average of 42 per month. During the fall and winter seasons, the average incidence was 110 per month.—*Anim. Dis. Trends, Aug., 1957.*

Bovine Pasteurellosis in Asia.—In Ceylon, where pasteurellosis had caused only a few deaths annually in cattle and buffalo prior to 1954, it has become epizootic, probably because of excessive rains and the increased number of artificial drinking ponds.

It is characterized by sudden onset, high fever, loss of appetite, cessation of rumination, frothy salivation, often lacrimation and a mucous nasal discharge, and high mortality. There is usually a marked edematous swelling of the throat which often extends to the cheeks and tongue and to the dewlap, and is invariably followed by a painful cough and labored respiration.

At necropsy, there is marked hyperemia of the internal organs, petechiae on the serous and mucous membranes, swelling and reddish brown discoloration of the tongue, and the lungs show necrotic inflammation in some and hyperemia and edema in other cases.

Losses usually stop, for several months, about a week following vaccination, using *Pasteurella multocida* type 1 (Roberts) vaccine.—*Ceylon Vet. J. (March-June, 1957): 24.*

[The strain of Pasteurella organism responsible for this "Asiatic pasteurellosis" is believed not to be present in North America.—ED.]

Proteins in Wound Healing

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DESPITE ASEPSIS and meticulous care in their surgical techniques, veterinarians sometimes observe retarded healing and wound disruption. This may be due to a lack of proper protein nutrition both pre- and postoperatively. Proteins are so necessary for life that their importance in the injured would seem obvious; yet they often are not considered by the surgeon in the care of his patients.

Whereas fat can be synthesized from carbohydrates and carbohydrates from proteins, the animal organism can not fabricate protein from any foods other than protein or its digestion products, the peptides and amino acids.[2]

Proteins, besides supplying amino acids for hormones and enzymes, for energy and metabolism, are essential for tissue repair for which they are the sole source of sulfur and nitrogen.[16]

The frequent deprivation of proteins and caloric needs, wholly or partially, is, in general, viewed with complacency or considered inevitable. The supposition[4] that tissue and other reserves will supply safely the needs of the body has lulled the surgeon into a false security. It is only when there is complete wound dehiscense that attention may be focused on nutrition and especially on proteins.

It is common for patients to be in a state of subclinical hypoproteinemia when they are subjected to surgery. This condition can be partly overcome or alleviated both pre- and postoperatively by proper selection of diets or by parenteral injections. Many other factors are involved in wound healing,[1] but they are beyond the scope of this article.

PROTEIN LEVELS

The normal level of protein is from 7.0 to 7.5 Gm. per 100 cc. of blood.[18] When this level reaches 6.0 Gm. per 100 cc., wound healing is definitely retarded, and below 5.5 Gm. per 100 cc., 70 per cent of wounds will disrupt. Edema occurs, with death ensuing, when the protein level drops to 2.5 Gm. per 100 cc. of blood.

Dogs, man, and probably other animals require a protein intake of about 0.454 Gm. per pound of body weight per day for maintenance of body proteins. However, a daily maintenance intake is insufficient for the rapid repletion of wasted tissues. A minimum of about 1 Gm. of proteins per pound of body weight per day should be given to establish a positive nitrogen balance and to promote rapid recovery of the patient following hypoproteinism. This excess protein insures a positive nitrogen balance, with ample proteins for daily maintenance and the repair of tissue and also for the replacement of proteins lost in wound exudates, etc.[3,5,9] It is highly desirable to administer sufficient dextrose with protein hydrolysates for the protein sparing action; *i.e.*, the dextrose can supply the energy needed by the tissues, and the protein hydrolysates can be utilized in tissue repair.[13]

It has been estimated that about twice as much dextrose (2 Gm./lb.) as protein hydrolysates should be provided as a minimum to stimulate tissue repair.[9]

HOW PROTEINS AID WOUND HEALING

Proteins are important in the osmotic relations between intracellular and extracellular fluids. The metabolism of proteins is in many ways more complicated than that of water, electrolytes, and glucose, and this should be understood before attempting effective parenteral correction of protein deficiencies. Recent studies reveal the concept of dynamic equilibrium which truly defines protein metabolism. This displaces the idea of static reserves of proteins. Proteins are in a steady state of rapid flux, that is, peptide bonds in the protein molecule are opening and closing[5] and certain amino acids are able to transfer a part of their nitrogen to other amino acids. This transfer is governed by the action of specific enzymes.

It is interesting to speculate on the probable influence of these newer concepts

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on the clinical problem. If a severe blood loss is encountered, the blood volume is spontaneously replaced from fluid in the body. Unfortunately, this fluid contains practically no proteins, so addition of fluid to the circulating blood produces both hypoproteinemia and anemia, which is the reason that its compensatory effect is inadequate. True enough, plasma proteins are regenerated eventually, but without outside aid this requires days instead of hours. According to one classification, there are three compartments of the body and proteins are limited to two of them—the circulating blood and the interior of the body cells. In the third compartment, which lies between the other two, *i.e.*, between the capillary wall and the cell wall, is the large volume of interstitial fluid which contains little, if any, protein except that which is in the lumen of the lymphatics. The proteins in the plasma, while quantitatively small, are extremely important physiologically in surgical patients, whereas the tremendous amount of proteins within the muscle cells have much less functional value. Plasma contains a number of proteins, namely, albumin, globulin, fibrinogen, and thrombin. For the purpose of protein alimentation, the albumin fraction is the one of greatest significance because: (1) In most surgical diseases, it is the fraction that is characteristically affected, and (2) its function is that of maintaining 85 per cent of the colloidal osmotic pressure of the blood.[5] Because of the importance of albumin in maintaining colloidal osmotic pressure and because it is the fraction that is depleted so frequently, the term "hypoalbuminemia" rather than the general term, "hypoproteinemia," should be used in describing most plasma protein deficiencies of interest to the surgeon.[6]

Hypoalbuminemia.—This may occur by any of the four following mechanisms.[6]

1) *Nutritional Hypoalbuminemia.*—This is a result of malnutrition, and is undoubtedly the most prevalent since it occurs in animals unable to eat sufficient proteins as well as those suffering from inadequate digestion or absorption of food. Experimental evidence shows that depletion of the albumin fraction begins immediately upon withdrawal of proteins from the diet.[7] It is a mistake, therefore, to rely upon stores of tissue proteins to protect the plasma. The longer the proteins are withheld, the more severe the hypoalbuminemia; if severe enough, nutritional edema eventually results and affects the healing mechanism. This is discussed later.

2) *Excessive Loss of Plasma Proteins.*—Hypoalbuminemia may result from any loss of plasma which is so rapid or extensive that the body can not regenerate or correct the loss quickly enough. A notable example of this is in acute injury associated with the exudation of plasma or blood into a large damaged area.

3) *Excessive Loss of Nitrogen.*—Normally, a few grams of nitrogen are lost each day in the urine, largely from tissue and plasma proteins used up in metabolic wear and tear. Certain diseases cause excessive destruction of plasma proteins which is revealed by large losses of nitrogen in the urine. Marked losses of nitrogen in the urine have been observed after operations as well as various traumas, especially fractures. The maximum effect is reached from the fourth to the eighth day after surgery or trauma.

4) *Defect in Albumin Synthesis.*—In hepatic diseases, hypoalbuminemia is frequent.[15] That the mechanism is due in large measure to hepatic insufficiency is based on the theory that the liver is the site of albumin synthesis.[12] Hence, the primary fault lies in the inability of the liver to utilize amino acids for the manufacture of plasma albumin.

Delayed wound healing is one of the more apparent manifestations of hypoalbuminemia in the body; the others are beyond the scope of this article.[6]

EXPERIMENTAL EVIDENCE

That a deficiency of proteins plays an important part in wound disruption was shown with dogs which were rendered hypoproteinemic by diets deficient in proteins; also by plasmapheresis.[20] An incision was made on each side of the linea alba through the skin, fascia, rectus muscle, and peritoneum. The size and location of the incision was the same in each dog. The incision on one side was closed with fine interrupted silk sutures, while No. 1 chromic 20-day catgut was used on the other side. Either disruption of the wounds or failure to heal occurred in 8 of 11 such dogs. Biopsy sections were made and the

tissues were found to be edematous with only a few fibroblasts in the area between the surfaces of the wound. In those sutured with silk, the edges were in apposition without any evidence of an attempt at union. In those sutured with catgut, the knot was often the only remnant, thus indicating a faster deterioration of catgut suture material in hypoproteinemic patients. Serious infection occurred in some of the wounds. The over-all picture was one of marked delay in tissue repair.[20]

In a similar study with hypoproteinemic dogs, attempts were made to prevent delayed wound healing and disruption by intravenous injections of acacia.[19] The theory seemed to be that the acacia would permit the osmotic pressure of the serum to remain at a higher level, and thus prevent edema of the tissues so that serum proteins could be withdrawn from the blood to be used in fibroplasia of the healing wound. However, the use of acacia in hypoproteinemic patients was not recommended.

In another study,[8] an abdominal wound was made in 53 rats, some of which were on a standard diet and some on a diet high in proteins. Six were killed daily, and the tensile strength of the healing wounds was tested. The latent period preceding the initiation of growth in the healing wound was not affected by a diet high in proteins but, once growth had started, its rate was distinctly increased by the diet high in proteins and the maximum strength was reached some two days earlier.

In other work with rats,[10] the wound strength on the fifth postoperative day was three times as great in those on a normal diet as in those on a diet low in proteins. The additional strength is due to: (1) a more rapid appearance and maturity of fibroblasts in the wound area, and (2) a more rapid maturation of the reticulum of the wound into mature collagen. Since sulfur is a necessary constituent of collagen, some workers feel that diets containing amino acids such as methionine and cysteine, which contain sulfur, are as effective in wound healing as a normal protein diet.[17,21]

In a study made at a surgical clinic, wound disruption occurred in 40 of 1,358 human patients.[20] The serum proteins were found to be significantly lowered in these patients.

ROUTES OF ADMINISTRATION

The oral route, where possible, is preferred over the intravenous route of administration since it results in a better utilization of the proteins. Only by the oral route can large amounts of the protein hydrolysates be administered satisfactorily.[9]

Parenteral protein therapy consists essentially of the intravenous administration of blood, plasma, albumin, and various casein hydrolysates. The casein hydrolysates may be given subcutaneously or intraperitoneally, or both, but if the need is urgent, they are given intravenously.

SOURCES

While temporary therapy with protein concentrates of various types is often valuable and necessary, only a highly nutritious diet over a long period will give satisfactory results.

From a dietary standpoint for carnivores, the best sources of proteins in the order mentioned are: soft-cooked whole eggs, lactalbumin from milk, casein from milk, chicken viscera, fish viscera, meat scraps (two thirds horsemeat and one third beef or hog liver), and then muscle meat.[14]

Whether dealing with carnivorous, herbivorous, or omnivorous animals, the end products needed are the amino acids; adequate proteins, capable of being converted to, or supplying directly, the needed amino acids, must be fed. However, animals unable to take foods by the preferred oral route may be given blood, plasma, albumin, and protein hydrolysates parenterally.

The administration of blood is essential at times for surgical success, but it should not be considered that this therapy will of itself remedy a deficiency of proteins. Adequate protein dosage, therefore, can not be approached by means of blood transfusions even if one were willing to accept the risk of hemoconcentration and polycythemia. It is used preoperatively chiefly because it rapidly replaces hemoglobin.[2]

Plasma and albumin solutions given intravenously are poor sources of proteins for nutritional needs; they are not incorporated into tissue proteins for five to seven days.[2] The plasma proteins must be broken down into amino acids before being utilized and they do not yield amino acids in correct proportions for utilization by the tissues. In fact, some essential amino acids

are not present in plasma proteins or blood.[9]

The protein hydrolysates are adequate sources of proteins—much more so than blood, albumin, or plasma—and are well tolerated both intravenously and orally. Most protein hydrolysates contain an unidentified fraction which improves the nitrogen retention, thereby making more proteins available for tissue building.

CONCLUSIONS

1) Even though hypoproteinemia is frequently encountered, veterinarians as a group seem unaware of the value of protein nutrition as an aid in wound healing.

2) Whereas fat can be synthesized from carbohydrates, and carbohydrates from proteins, the animal organism can fabricate proteins only from protein material.

3) A protein intake of 0.454 Gm. per pound of body weight per day is required in animals for normal function. To insure a positive nitrogen balance, a minimum of 1 Gm. per pound should be given; dextrose (2 Gm./lb./day) should also be given for energy and protein sparing action, to allow the proteins to be used in tissue repair and synthesis.

4) Protein metabolism is in a constant state of flux. Proteins aid largely in maintaining osmotic pressure between intracellular and extracellular fluids. The body is slow in replacing protein losses from its own tissues.

Albumin alone accounts for 85 per cent of the colloidal osmotic pressure of the plasma; therefore, it is the most important plasma fraction in wound healing.

Hypoalbuminemia is present in (1) malnutrition, (2) excessive loss of plasma proteins, (3) excessive loss of nitrogen in body metabolism, and (4) defect in albumin synthesis. One of the most apparent manifestations of hypoalbuminemia is delayed wound healing.

5) Experiments show that the maximum strength of the healing wounds was reached two days earlier in animals on a high protein as compared to the low protein diet. Wound disruption is also common in animals suffering from hypoproteinemia. High protein levels prevent tissue edema and promote increased fibroplasia, giving wound strength three times as great as low protein levels on the fifth postoperative day.

6) The oral route is preferred and is the only route by which large amounts of protein materials may be given. When they can not be given orally, the parenteral route used is determined by the need of the patient.

7) For parenteral administration, the protein hydrolysates should be used since the essential amino acids are present and readily available for tissue synthesis.

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Wheat Pasture Poisoning in Cows.—This condition, which had been reported from Texas and Oklahoma, especially during periods of plentiful moisture, was observed only in mature cows in late pregnancy or those nursing a young calf. The affected cows showed excitement, incoordination, and loss of appetite; then viciousness, muscular twitching, grinding of the teeth, profuse salivation, and prominence of the nictitating membrane as in tetanus. If untreated, convulsions, coma, and death sometimes occurred in six to ten hours. The blood serum content of inorganic phosphate, calcium, and magnesium was usually decreased. The best treatment seemed to be the injection of calcium gluconate solution intravenously or preferably intraperitoneally, and removal from the wheat pasture for a time. Recurrence has not been observed in recovered animals.—*F. H. Sims and H. R. Crookshank in Southwestern Vet. (Summer, 1957): 277.*

Nitrate Poisoning of Cattle.—Deaths of dairy cows on three farms, in New Zealand, were found to be due to ingestion of plants containing toxic amounts of nitrate, *i.e.*, immature turnip tops, a weed (*Amaranthus retroflexus*), and immature green oats. The toxicity of the first two was established by feeding trials. Most of the deaths occurred suddenly without previous clinical signs. Prompt treatment with intravenous methylene blue saved some cows. A cow can consume a toxic amount of such plants in one hour.—*New Zealand Vet. J. (June, 1957): 51.*

Value of Enzymes in Feed for Young Pigs.—Pepsin and pancreatic amylase are present in insufficient quantities to digest vegetable-based feeds until pigs are 4 to 5 weeks old. This is not a problem when pigs are weaned after that age. The results of tests when pigs are fed under laboratory conditions vary, probably because under relatively sterile conditions the food remains in the alimentary tract longer, allowing better digestion even with a shortage of enzymes. Pepsin, which is extracted from the gastric mucosa of swine, has been produced commercially for over 70 years. If early weaning of pigs were practiced extensively, it is estimated that perhaps 3,000 tons of pepsin would be needed annually.—*J. Agric. and Food Chem. (July, 1957): 484.*

Pantothenic Acid in Swine Nutrition.—Weanling pigs fed a ration of natural feedstuffs low in pantothenic acid showed poor gain, diarrhea, and colitis. The large intestines showed congestion, edema, superficial discrete ulcers, and excessive mucus, all of which could be prevented by feeding adequate pantothenic acid. The deficiency signs also included incoordination and "goose-stepping." Whereas about 12.5 mg. of calcium pantothenate per kilogram of solid appeared to be required, a basal ration of 82 per cent yellow corn, 16 per cent soybean meal, and mineral supplements was found to contain 6.7 mg. per kilogram. In some experiments, feeding chlortetracycline seemed to have a pantothenic acid-saving effect.—*Nutr. Rev. (Sept., 1957): 275.*

Pantothenic Acid Deficiency.—Calves fed a pantothenic-acid free synthetic milk diet developed rough coats, dermatitis under the lower jaw, excessive nasal mucus, loss of appetite, reduced growth rate, and loss of weight, followed by death. The lesions included edema, pneumonic changes, demyelination of sciatic nerve and spinal cord, and softening and congestion of the cerebrum. Affected calves responded to calcium pantothenic therapy. (Digestion in young ruminants resembles that in nonruminants which need B vitamins.)—*J. Nutr. (Feb. 10, 1957): 195.*

Gibberellic acid, the new plant growth stimulator, can turn grass green in winter but whether it will lengthen the pasture season remains to be seen. Its effect on the growth and quality of alfalfa, especially its carotene content, has not yet been determined.—*Feeds Illus. (July, 1957): 12.*

Editorial

Richard Shope, Benefactor of Animals

On May 8, 1957, the Association of American Physicians awarded the Kober Medal to Dr. Richard E. Shope, the second time this award has been given to a physician for contributions to the health of domesticated animals. The previous recipient was Dr. Theobald Smith.

Dr. Richard E. Shope

Excerpts of the remarks of Dr. Peyton Rous, famed pathologist and authority on cancer, who made the award, and of Dr. Shope are presented here because of their interest to humanity in general, but particularly to veterinarians.

Dr. Rous mentioned that Dr. Shope's father was an overworked doctor, his mother a former teacher, and that the "most vital part of his education . . . went on at the family farm just outside of Des Moines." There, Shope raised pigs,—"the beginning of his deep love for swine, and also ran a milk route." However, when dressing chickens, "he got so interested in their insides as to slack on the job," so that task went to another. Shope was "really apprenticed to the ways of animals."

After Dr. Shope received his M.D. degree at the University of Iowa, in 1924, and served there for a year as an instructor of pharmacology, he joined the staff of the Department of Animal Pathology of the Rockefeller Institute. There, he first worked on tuberculosis and on the cholesterol and sugar of blood and colostrum.

However, according to Dr. Rous, Shope had a tendency to "venture on his own," as when he suspected, then proved, that "house flies, by reason of their dissolute way of life," teemed with bacteriophages. Next he "heeded the call of the pig, and . . . found in cholera a leucopenia" of diagnostic significance.

Working with swine influenza, Shope "transmitted the disease to other pigs . . . isolated a bacillus . . . then obtained a virus as well." He then (1931) suggested "that Pfeiffer's bacillus and a filterable virus might act in concert to cause human influenza."

Dr. Shope also found a "virus causing another, and truly terrible, disease which caused . . . cattle to bite and tear at themselves until they die, . . . mad itch it is called."

"In 1932, while hunting, he came upon a unique disease of cottontail rabbits . . . fibromas . . . due to a virus." The next year he investigated another disease of rabbits "causing huge warts and horns on their skin," — benign, keratinizing epidermal papillomas, which also yielded a virus.

Shope now had a total of "four remarkable virus diseases," and continued to work with three of them. "What he really wanted to find was the source of the viruses and in two cases he found it."

He proved mad itch to be "identical with pseudorabies of Hungarian cattle. It killed every cow attacked, yet . . . wasn't catching." In pigs, it was highly contagious but mild, and all cattle affected "had been with pigs." But how was the virus distributed? "Pigs don't visit from one farm to another. But rats will . . . so Shope inoculated rats, . . . and the pigs that ate the dead rats fell sick." He still "wants to know how rats get" the virus.

In infectious fibromatosis of rabbits, he observed some similarities to the fatal infectious myxomatosis of South America. He found that animals in which the fibromatosis had run its course were immune to myxomatosis." Thus, the "Shope" vaccine for myxomatosis.

The fourth disease, infectious papilloma-

tosis of rabbits is still being studied whenever there is cancer research.

In 1941, Commander Shope "was summoned . . . to produce a vaccine against rinderpest," a secret joint enterprise with Canada, on Grosse Isle in the St. Lawrence River below Quebec. In 19 months his group developed a vaccine (*Am. J. Vet. Res.*, April (Part 2), 1946), that has been used "on millions of cattle . . . and has only lately been superseded."

Later, while on duty at a navy laboratory on Guam, Dr. Shope found a mold, an extract of which "neutralized the virus of swine influenza" but which slowly lost its effectiveness.

Dr. Shope now serves on 17 important boards and committees; one is the Grayson Foundation for study of race horse diseases. He is "beyond all else a naturalist . . . [his] feeling for animals is not sentimental but compassionate . . . to watch how he handles them is a delight. . . ."

"In all of his major discoveries he has been sole discoverer. . . . As a go-it-aloner he somehow manages . . . huge tasks. Shope does not concern himself with the intimate relationships of viruses to cells, but with their gross effects, and whence they came, whither go, and the secret haunts in which they lie up between times. He is a virographer, . . . continually exploring new animal territory infested with viruses. . . ."

DR. SHOPE'S RESPONSE

In his response, Dr. Shope acknowledged the influence of individuals who "played a large role in . . . my scientific career." One was Dr. Theobald Smith, at Rockefeller Institute, who, "although possessed of great imagination and . . . uncanny foresight, was a meticulously methodical worker, who abhorred the waste of anything—time, material, or animals." Another was Dr. Paul A. Lewis, who later died of yellow fever while studying it in South America.

Both Lewis and Smith believed that "disease should be studied as it occurred under natural conditions; both had an aggressive distaste for laboratory gadgetry, and both believed strongly that . . . equipment . . . would not substitute for careful thought and close and accurate observation."

Dr. Shope then paid tribute to a veterinary practitioner, Dr. Fred J. Crow of Iowa City, Iowa, "who permitted me to spend . . . time with him in the field . . . and who guided me during my initial experience with swine influenza. . . He got me my first case of swine pox and . . . of pseudorabies and . . . served as the connecting link between the practicality of the field and the science of the laboratory."

EDITORIAL COMMENT

Of particular interest are Dr. Shope's remarks on studying disease "as it occurred under natural conditions." There is a tendency among some research workers to depreciate field observations, the conception seeming to be that research can be done only while peering into a microscope or while manipulating laboratory equipment. Was not Dr. Cooper Curtice contributing mightily to science when "examining ticks on a cow dead of Texas fever." (*See* fly leaf in Hagan's text, "Infectious Diseases of Domestic Animals.")

There is also significance in Dr. Rous' emphasis that Shope was "really apprenticed to the ways of animals," a qualification apt to be insufficiently considered by faculty committees when evaluating applicants for veterinary training. The shift of population from rural to urban residence makes it increasingly difficult for applicants to qualify, as Shope well could have done, with that "most vital . . . education . . . on the family farm," for which we believe there is no adequate substitute in veterinary training.

That all do not agree is indicated by a capable research worker who referred to the "ridiculous situation" wherein "farm or ranch background students are allowed some preference," or a farm "experience requirement must be met," even questioning whether such experience makes better rural practitioners. However, those who have observed the struggle of young urban-raised veterinarians trying to cope with rural practice problems, or the inability of urban-raised pathologists to picture, mentally, important problems relative to livestock diseases, know how some of them are handicapped.

And lastly, Dr. Shope's comments on Dr. Crow's aid should definitely remind us of the benefits of cooperation between field and laboratory workers.

Current Literature

ABSTRACTS

Bloat Therapy and Prophylaxis

Twenty-two beef cattle grazing alfalfa pasture were allotted to two groups and bloat-preventive treatments were alternated between groups. An alkyl aryl sodium sulfonate-type detergent (Ultrawet "K") was administered via gelatin capsules at rates of 20 and 30 Gm. daily per 1,000 lb. of body weight. At the higher rate, a significant reduction in incidence and severity of bloat occurred. During bloat, the ingesta volume increase, foam durability, and degree of foaminess of rumen ingesta were markedly increased, surface tension and viscosity were moderately increased, while the specific gravity and pH were lowered moderately. All these properties were altered in the opposite direction (toward prebloat levels) during detergent treatment. Trisodium phosphate, 30 Gm. daily, had little influence on bloat occurrence or on the measured rumen fluid properties. Grazing habits were not altered noticeably as a result of either detergent or trisodium phosphate treatment.

Bloat-provocative alfalfa juice was prepared and administered by stomach tube to young dairy animals. Neither cholesterol nor the detergent effectively reduced bloat or greatly altered the measured rumen fluid characteristics when added to the alfalfa juice before administration.—[*J. T. Blake, N. L. Jacobson, and R. S. Allen: Effectiveness of Various Measures in Bloat Therapy and Prophylaxis and the Resulting Influence on Rumen Fluid Characteristics. Am. J. Vet. Res., 18, (Oct., 1957): 756-760.*]

Infection of Mice with Salmonella Kentucky

Salmonella kentucky was isolated from the liver of a CFW albino mouse which presented macroscopic lesions typical of mouse typhoid.

Experimental infection of mice with this Salmonella sp. required inoculation (intraperitoneal, subcutaneous, or into foot pad); introduction by oral or conjunctival routes was unsuccessful.

It is concluded that *S. kentucky*, never hitherto isolated from mice, is unlikely to be a cause of epizootics of typhoid in mouse colonies.—[*Georges H. Werner: Infection of Laboratory Mice with Salmonella Kentucky. Am. J. Vet. Res., 18, (Oct., 1957): 954-955.*]

BOOKS AND REPORTS

Veterinary Pathology

This book covers the widest scope of any general text on veterinary pathology. It will serve as a general text for the student and as a reference for the pathologist, practitioner, research worker, and members of the various medical sciences. It includes accounts of the general, special, and systemic pathology of all domesticated animals (except poultry), laboratory animals and, to some extent, captive wild animals.

Of the 27 chapters, the first eight are devoted to the degenerative processes of disease. The next five deal with diseases caused by viruses and rickettsiae; simple bacteria; higher bacteria, fungi, and spirochetes; protozoa; and parasites. The next three chapters deal with radiation, poisons, and disorders of metabolism. The remaining eleven chapters discuss pathological changes in the various systems.

Those preparing this text were extremely fortunate in that, aside from their vast personal experience in the field, they had access to the world's greatest collection of pathological material upon which to base the information presented. Readers will appreciate the concise descriptions, the excellent gross and microscopic illustrations, and the selected reference list at the end of each chapter.—[*Veterinary Pathology. By H. A. Smith and T. C. Jones. 959 pages. Illustrated. Lea and Febiger, Washington Square, Philadelphia 6, Pa. 1957. Price $17.50.*]—WAYNE H. RISER.

Your Dog's Health Book

This book was written as a guide to general health information for the dog owner. The author was assisted by 29 veterinarians. The subject matter is concerned with housing, grooming, nutrition, training, and health problems. It is presented in language the average animal owner will understand and appreciate.—[*Your Dog's Health Book. Edited by Jack Denton Scott. 289 pages. Macmillan Co., 60 Fifth Ave., New York, N.Y. 1956. Price $5.00.*]—WAYNE H. RISER.

Year Book of Cancer

The purpose of this book is to provide abstracts of the most important articles on cancer in man which were published in 1956. The various kinds of cancer are discussed, along with methods of diagnosis, case reports, evaluation of treatments, and prognosis. There are also chapters on basic research, special surgical problems, and psychological aspects of the disease. The book is well supplemented with illustrations and tables.—[*Year Book of Cancer. Compiled and edited by Randolph Lee Clark, Jr., and Russell W. Cumley. 572 pages. Year Book Publishers, 200 E. Illinois St., Chicago. 1957. Price $7.50.*]

Human Cancer

The authors discuss the detection of cancer, its behavior, treatment, and biochemistry in the human patient. There are separate chapters on cancer in the various tissues, *i.e.*, lung and mediastinum, gastrointestinal tract, bone, skin, reproductive organs, soft tissues. At the end of each chapter, an extensive list of references is given.—[*Human Cancer, A Manual for Students and Physicians. By Maurice M. Black and Francis D. Speer. 273 pages. Illustrated. Year Book Publishers, Inc., 200 E. Illinois St., Chicago. 1957. Price $7.50.*]

THE NEWS

National Science Foundation Fellowships Announced

The National Science Foundation announced early in October the availability of fellowships in four programs for advanced study and research in the natural sciences and including mathematics, physics, medicine, biology, engineering, and other sciences for 1958-1959.

The four programs include predoctoral, postdoctoral, senior postdoctoral, and science faculty fellowships. Applications for the first two categories may be obtained from the Fellowship Office, National Academy of Sciences, 2101 Constitution Ave., N.W., Washington, 25, D. C., and for the latter two, from the Division of Scientific Personnel and Education, National Science Foundation, Washington 25, D.C.

The closing dates for receipt of applications are late in December, 1957.

Dr. Dearing Appointed Assistant Health Director, O.D.M.

Dr. W. Palmer Dearing, formerly Deputy Surgeon General, U. S. Public Health Service, was appointed on September 27 to the newly created post of assistant director for health in the Office of Defense Mobilization. The new position was established to better coordinate federal government health activities essential to national defense.

Dr. Dearing was a featured speaker at the AVMA convention in Detroit, in 1949, on the subject, "The Future Health Needs of the Nation."

AMONG THE STATES AND PROVINCES

Illinois

Mississippi Valley Association.—The Mississippi Valley V.M.A. held its annual meeting Oct. 23-24, 1957, at the Père Marquette Hotel, Peoria, Ill.

The following speakers and their subjects appeared on the program: Drs. A. C. Todd, University of Wisconsin—Parasitology in 1957 Veterinary Practice; L. E. St. Clair, University of Illinois—Applied Anatomy; C. L. McGinnis, practitioner, Peoria—Small Animals; C. A. Brandly, dean, University of Illinois—Duties of the Extension Veterinarians; Glynden T. Easley, Turner Ranch, Sulphur, Okla.—Beef Cattle; J. D. Ray, lab director, Affiliated Laboratory Corp.—Round-Table on Swine Diseases; and T. H. Brasmer, practitioner, Danville, Ill. —Canine Geriatrics.

s/WILLIAM L. BEER, *Secretary.*

Georgia

Dr. Joseph Tucker (BER '07) and his wife, Hedwig Zellner Tucker (left), Atlanta, at the fiftieth anniversary celebration on July 9, 1957, commemorating his half-century of practice as a graduate veterinarian.

Dr. Tucker, a German veterinarian, attended Alabama Polytechnic Institute, Auburn, after he was forced to leave his native country under Hitler's regime. Soon after, he opened an animal hospital in Atlanta.

Kansas

Dr. Earl J. Splitter Joins ARS.—Dr. Splitter (KSC '43), formerly with Kansas State College and an authority on eperythrozoonosis in swine, is now with the State Experiment Stations Divisions, Agricultural Research Service, U.S.D.A., Washington, D. C.

New Mexico

Women's Association.—The annual meeting of the Women's Auxiliary to the New Mexico V.M.A. was held at the Fez Club, Sept. 9, 1957.

Mrs. Gladys Evans, Albuquerque, presented a brief resume on the 1956 and 1957 annual meetings of the AVMA Women's Auxiliary. It was voted to continue contributions to the AVMA Research Fund.

New Officers for the coming year are: Mrs. Pauline Wiseman, Roswell, president; Mrs. Katherine Heise, Albuquerque, vice-president; Mrs. Shirley Payne, Albuquerque, secretary-treasurer; and Mrs. Gladys Evans, national delegate.

s/MRS. EARL L. PAYNE, *Secretary.*

Oregon

Portland Association.—The Portland V.M.A., at their Sept. 10, 1957, meeting, elected the following officers for the ensuing year: Drs. Glenn B. Burleigh, Portland, president; Donald H. Holtzheimer, Gresham, vice-president; and Donald L. Moyer, Portland, secretary-treasurer.

s/DONALD L. MOYER, *Secretary-Treasurer.*

Washington

Women's Auxiliary.—The annual meeting of the Women's Auxiliary to the Washington V.M.A. was held in Longview, Sept. 9-10, 1957.

The Auxiliary supported the paid executive-secretary plan that was initiated by the Washington State V.M.A. this year. It was voted to send $50 to the Washington State Library Fund, $50 to the National Student Loan Fund, and a lesser amount to the Research Fund.

A special memorial contribution was made also in honor of Dr. George Staggs, Tacoma, one of the oldest members of the Association, who died recently.

The new officers elected for the ensuing year are: Mrs. Don W. Clarke, Seattle, president; Mrs. Ernest C. Stone, Pullman, president-elect; Mrs. Phillip S. Millard, Everett, secretary; and Mrs. Robert E. Ebright, treasurer.

The delegate to the house of representatives of the Women's Auxiliary to the AVMA is Mrs. Don W. Clarke and the alternate delegate to the convention is Mrs. T. Robert Phelps, Vancouver.

s/MRS. FRED C. CUMMINGS, *Secretary.*

U. S. GOVERNMENT

Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U.S.D.A. are reported as of Sept. 30, and Oct. 2, and 3, 1957.

TRANSFERS

George E. Anderson, from Moultrie, Ga., to Dothan, Ala.
Edward C. Arthur, from Indianapolis, Ind., to Philadelphia, Pa.
Woodrow W. Harkins, from Lansing Mich., to Jacksonville, Fla.
William H. Irvin, from Chicago, Ill., to Indianapolis, Ind.
Mathias J. Kemen, Jr., from Montpelier, Vt., to Indianapolis, Ind.
George Migaki, from Portland, Ore., to Beltsville, Md.
Dean A. Price, from Boise, Idaho, to Cheyenne, Wyo.
Clifford W. Wilder, from Albany, N.Y., to Harrisburg, Pa.
Don R. Wilson, from Dothan, Ala., to San Antonio, Texas.

RETIREMENTS

Jesse R. Farr, Augusta, Ga.
Luna O. Lietzman, Anaheim, Calif.
Harper H. Shearer, Chattanooga, Tenn.

DEATHS

Alva A. Johnson, Indianapolis, Ind.
Sidney A. Steiner, Philadelphia, Pa.

STATE BOARD EXAMINATIONS

COLORADO—Jan. 15-16, 1958, Arvada; and first week in June, depending on graduation date, Fort Collins. W. D. Stauffer, secretary, 5500 Wadsworth Blvd., Arvada, Colo.

ILLINOIS—Dec. 4-6, 1957, Chicago. F. B. Selcke, registration superintendent, Springfield, Ill.

MINNESOTA—Jan. 6-7, 1958, St. Paul. A. C. Spannaus, secretary, Route 1, Waconia, Minn.

NEW JERSEY—Dec. 27-28, 1957, Trenton. Joseph A. S. Millar, secretary, Box 172, Deal, N.J.

NORTH DAKOTA—April 9-10, 1958, Fargo. M. C. Hawn, secretary-treasurer, 1407 13 St. N., Fargo, N. Dak.

NEW MEXICO—Jan. 13, 1958, Santa Fe. Edwin J. Smith, secretary, P.O. Box 4385, Santa Fe, N.M.

TEXAS—Jan. 15-17, 1958, Austin. T. D. Weaver, executive secretary, 207 Capital National Bank Building Austin 16, Texas.

DEATHS

Star indicates member of AVMA

Walter S. Davis (CIN '18), 61, Bladen, Ohio, died Aug. 18, 1957. He had been troubled with a heart condition for some time.

Dr. Davis had been the past-master of the Gallia Masonic Lodge 469 at Bladen; he was a World War I veteran.

Surviving are his widow, Rena Lewis Davis, and seven children.

★**Warren J. Embree** (CVC '10), 69, Columbus, Ohio, died late in August, 1957. The former, and retired, chief veterinarian of the Webster Weighing and Inspection Bureau of the New York Central Railroad was widely known in livestock conservation circles and took an active part in that work for many years.

Dr. Embree is survived by his widow and a sister.

Earl A. Hays (STJ '18), 63, Fort Dodge, Iowa, died at the family summer home at Okoboji, Aug. 12, 1957. He had been in ill health for several years and apparently suffered a heart attack.

Dr. Hays is survived by his widow, a son, and three grandchildren.

★**Frank P. Mathews** (COL '23), 65, Arroyo Grande, Calif., died Aug. 1, 1957, after a long illness.

Dr. Mathews served in World War I from 1917 to 1919. After graduating from Colorado A. & M. College in 1923, he continued his education until he received his Ph.D. from the University of Michigan in 1937. Ill health caused him to move to Santa Maria, Calif., in 1947.

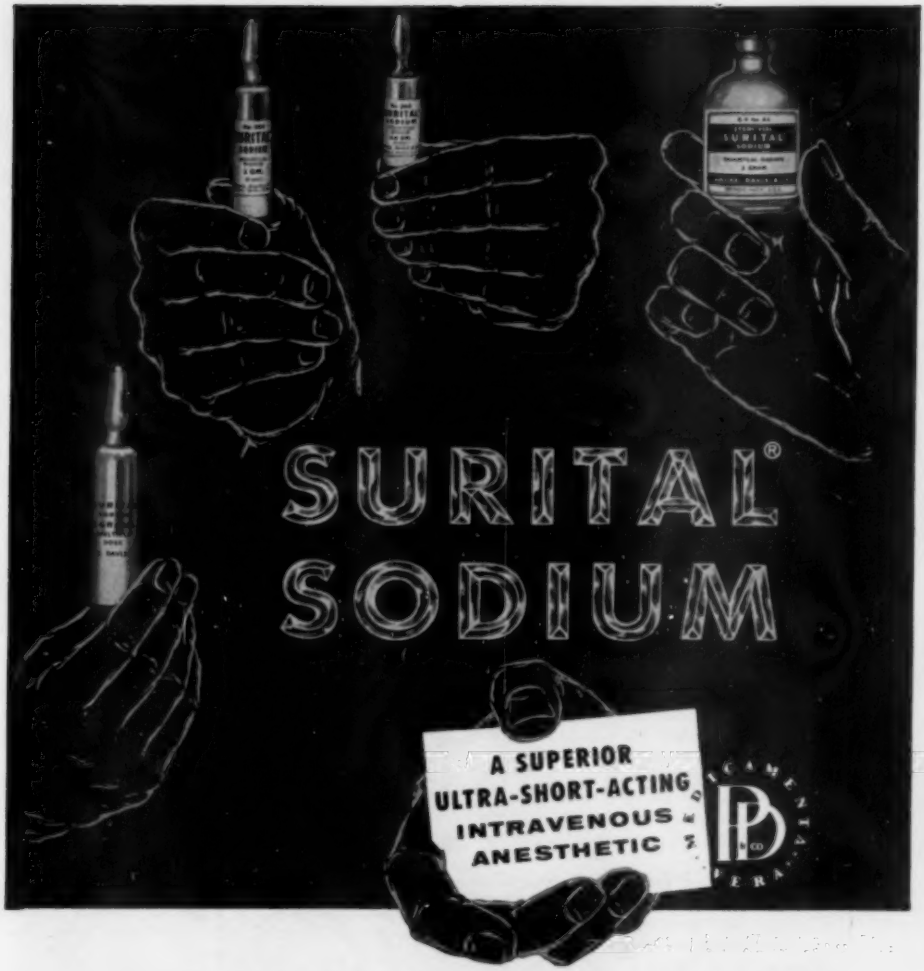
He is survived by his widow, Genevieve, one son, and two grandchildren.

★**John Saprausk** (LAT '30), 53, died July 29, 1957, in Pomona, Calif., following a heart attack.

Dr. Saprausk was born in Latvia in 1903. Following graduation from the University of Latvia, Riga, he was engaged in general veterinary practice there until 1936. He served as chief veterinarian of the district of Riga, Latvia, from 1937 to 1944. Forced to leave his native country in 1944, he went to Germany and completed work on a thesis at the College of Veterinary Medicine in Hanover for the D.M.-V. degree.

In 1947, Dr. Saprausk came to the United States and settled in California.

He is survived by his widow, Irma.



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PROFESSIONAL LITERATURE AVAILABLE ON REQUEST

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Report of the AVMA Representative to the Annual Meeting of the National Association of Artificial Breeders

Mr. Chairman, President Kester, Members of the AVMA, and Guests:

It is my distinguished pleasure to report from the artificial insemination industry and the tenth annual meeting of the National Association of Artificial Breeders. Some 760 enthusiastic supporters of the philosophy stamped on their national emblem, "Better Cattle for Better Living," gathered at the Royal York Hotel in Toronto on August 11 to 14.

The Association claims an increase in membership this past year, bringing their total to 90 member organizations, 77 within the United States representing 5,750,148 cows enrolled in the artificial breeding program. Only 12,000 cows, serviced by seven small organizations, are not included on the roster of this organization.

J. Stanley Earl, president of the New York Artificial Breeders Cooperative, was re-elected president for his fourth term. Elected directors who assist President Earl and Harry Herman, executive secretary, are: Richard Kellogg, general manager, Central Ohio Breeders Cooperative, vice-president; Charles Krumm, manager, Southern Wisconsin Breeders Cooperative at Madison; Lantz Wornack, president, Louisiana Artificial Breeders Cooperative at Winnsboro; Jay C. Harris, manager, Northwest Breeders Cooperative, Burlington, Wash.; Paul Heller, manager, Connecticut Artificial Breeding Association at Woodbridge; and Frank Heim, director, Michigan Artificial Breeders Cooperative at Lansing. These seven men, four of whom are managers and three directors of the largest and most progressive studs in the country, have a tremendous responsibility to the dairy industry and indirectly to the agricultural industry of America.

Progress in research on problems dealing with reproduction, genetics, and semen quality measurements and storage has been satisfying. Last year, $15,678.03 was allocated to research by this association. Fifteen active projects are being supported; $4,480.00 was allocated to research on vibriosis and cooperative research with member organizations made an additional $10,000 value available for study on this costly disease.

We of the veterinary profession should be cognizant of this effort by the Research Committee of the N.A.A.B. and offer any assistance possible, such as monetary contributions, personnel, clinical data, or cooperative research through our own research council activities. The chairman of the N.A.A.B. Research Committee is Executive Secretary Harry Herman. Members of this committee are Dr. T. E. Patrick, Louisiana; Dr. R. J. McDonald, Woodstock, Ont.; Dr. Henry Dunn, Cornell University; Dr. W. H. Dreher, Wisconsin; and F. G. Stevenson, Missouri.

Our Research Council members and other committees of the AVMA would do well to keep in contact with this N.A.A.B. group, in order to further our common goals relative to disease diagnosis, control, and eradication.

The N.A.A.B. is seriously concerned with the possible effects upon the cattle industry of undesirable genetic factors. Members are joining with the Purebred Dairy Cattle Association in an effort to develop means of detecting carriers and eliminating these animals quickly. Veterinary practitioners, educators, and researchers should be conscious of this effort and report any incidence of such inherited defects as hairlessness, single toe, fused teats, flexed pastern, taillessness, abnormal mucosal lining of the digestive tract, prolonged gestation, and the failure of pituitary gland development in unborn calves. This latter defect, reported recently by California researchers, has been found only in Guernsey cattle and causes retention of the fetus for abnormally long periods, extending from 292 to 526 days after service, with an average of 401 days. Calves cease to grow at about the seventh-month stage, and birth is delayed usually until the calf dies. Caesarian section calves lived only a few minutes. This differs from prolonged gestation as reported in Holstein-Friesians. In the latter case, the fetus grows larger and larger.

Studies relative to semen diluters have yielded two important discoveries. Almquist and associates at Pennsylvania have reported on the superior conception rate obtained with nonfrozen semen extended in a milk-glycerol solution. These workers also have strong evidence to indicate that semen can be collected twice weekly with no adverse effects on the bulls, rather than once weekly, as practiced in most studs. This could double the potential of some great sires.

Van Demark and co-workers at the University of Illinois have developed a method of putting sperm cells "to sleep" in a special formula diluter containing carbon dioxide gas. The semen is stored at room temperature (65 to 80 F.). Some samples of sperm cells have remained viable for a month, while 7-day-old semen has resulted in a 75 per cent conception rate. Frozen semen research continues at a tremendous pace. Newer, more practical, and more efficient methods of transporting and storing frozen ampules in the field are being developed. Liquid air or liquid nitrogen, in specially designed containers, is an effective method of storing semen from many different bulls in a relatively small area at temperatures low enough to offer an ample safety margin. Mechanical units are more efficient and becoming more in line with cost of other equipment in refrigeration field.

These facts have opened a tremendous new avenue of artificial breeding—beef cattle insemination. It is inevitable that more and more men in the beef industry will avail themselves of the great advantages offered by frozen semen techniques. In a recent issue (September, 1957) of the western

Dr. Hill is associate professor of Clinics and Surgery, Veterinarian in Charge, Artificial Breeding and Bull Testing Service, Colorado State University, Fort Collins.

proven results in these diseases of small animals

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REFERENCES: 1. Mosier, J. E.: Vet. M. 50:605 (Nov.) 1955. 2. Bolloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Pollock, S.: J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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edition of *the Aberdeen-Angus Journal*, a prominent educator, M. E. Ensminger, chairman of the Department of Animal Husbandry at the State College of Washington, states: "Artificial insemination is here to stay, and we (the beef breed associations) should not attempt to legislate against it. I think it is a sad mistake to pass rules to the effect that the semen dies with the sire. I predict that someday semen from these 'King Tuts' will be respected and used as long as possible. Such is progress."

Mr. Ensminger is correct; progress will dictate that the beef industry avail itself of this powerful tool for livestock improvement.

With the increased interest in breeding commercial cows to progeny-tested and performance-tested sires, the beef industry will find themselves searching for means of extending the use of such scarce and valuable bulls, just as the dairy industry did when the demand for proved sires became so great in the wake of a rapidly growing artificial breeding business.

Veterinarians should re-assess our contributions in the past to artificial breeding, reproductive disease control, sterility practice, and pregnancy examination, and take a long look at the future.

In addition to the potential in the beef cattle industry, interest has been revived in artificial breeding of swine, goats, sheep, and horses in all stratas of society. Improved equipment and refined techniques make artificial insemination of these species a practical and valuable veterinary service.

Dr. C. F. Huffman, president of the American Dairy Science Association, reminds us that milk alone pours about $4.7 billion into the pockets of American dairymen. This product, plus the large supply of veal, in addition to 20 per cent of our edible beef, make dairying account for 20 per cent of our gross national agricultural income. Surely no other single phase of livestock production will displace dairying in numbers of animals artificially inseminated, but a great new field in related physiology of reproduction, involving all species of animals, is being opened by the success and economical impact of 20 years of inseminating the dairy cow.

In my personal opinion, the progress of the N.A.A.B. has only one direction in which to go, and that is forward to greater numbers of members, greater number of cows being inseminated, to better and more carefully selected sires, and to more rewarding results in self-initiated research. Quoting (and by so doing I endorse this quote), Dr. J. M. McLean, manager of Quinte District Cattle Breeding Association, Belleville, Ont., writes in the *Canadian Journal of Comparative Medicine and Veterinary Science* (June, 1957): "Because artificial insemination today is a part of our national economy, we, as veterinarians, must continue to remain an active part of the organization."

An efficient level of animal reproduction, with constant improvement in the resulting offspring, is absolutely essential if we are to assure our posterity of three square meals a day in a population-burdened world to come. The ultimate goals of the AVMA and the N.A.A.B. are identical; therefore, our efforts must be inseparable.

Respectfully submitted,
H. J. Hill, D.V.M.

APPLICATIONS

Applicants—Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and names of his endorsers.

Second Listing

BOFILL, JORGE, Maruri 1471, Santiago, Chile.

BOWIE, DWAIN T., 76 Med. Vet. (VFIL) APO 230 c/o P.O., New York, N.Y.

DALZIEL, GEORGE T., 834 Robinhood Lane, La Grange Park, Ill.

LICANCURA, ALEJANDRO LENAM, Casilla 4, Nueva Imperial, Chile.

LYLES, D. I., P. O. Box 788, Tuskegee Institute, Ala.

SALVADOR, BAZ, Ave. Marti, No. 61-14, Mexico D. F., Mexico City, Mex.

WEBB, ALFREDA J., P. O. Box 676, Tuskegee Institute, Ala.

WEIL, FREDERICK, 1611 U.S.A.F. Dispensary, McGuire AFB, N. J.

WILLIAMS, RAYMOND C., P. O. Box 267, Tuskegee Institute, Ala.

WILLIAMS, THEODORE S., P. O. Box 216, Tuskegee Institute, Ala.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

Second Listings

University of Illinois

CLARK, LAWRENCE G., D.V.M., 12 Lincoln Way, Sparks, Nev.

COSTER, RICHARD D., D.V.M., Rt. 1, Box 35, Elgin, Ill.

University of Minnesota

HANSEN, MICHAEL H., D.V.M., Rockville, Minn.

University of Montreal

L'ECUYER, CONRAD, D.V.M., 18 Perrot St., Wrightville-Hull, Que.

Texas A. & M. College

FOWLER, JACK L., D.V.M., Rt. 1, Lakeview, Texas.

WILLIAMS, EUGENE H., D.V.M., 9721 Wallisville Rd., Houston, Texas.

WOMEN'S AUXILIARY

President—Mrs. Leslie H. Moe, 1814 W. Third Ave., Stillwater, Okla.
Secretary—Mrs. A. W. Eivers, 1595 N. 18th, Salem, Ore.

Auxiliary Affiliates.—An auxiliary affiliated with the Women's Auxiliary to the American Veterinary Medical Association may be a state, provincial, or regional organization. State and provincial groups are allied with constituent veterinary medical associations of the AVMA. There are 45 state and three provincial affiliates. Regional auxiliaries are auxiliaries to nonconstituent associations, and may be associated with a local, city, county, or regional group. These are organized groups of women with an interest in the veterinary profession. There are now 25 such affiliates.

At present, the only requirement for affiliation is that the auxiliary be organized and all officers, delegate and alternate, are members of the Women's Auxiliary to the AVMA. A group interested in affiliation is usually more than just a social bridge club. The affiliates are invited to support the national projects but it is not mandatory. It is more effective to administer projects at the AVMA auxiliary level, which are national in scope, such as the Student Loan Fund, awards, and the student auxiliaries.

In the last few years, three important committees have been appointed which may be of direct help to the affiliates, if so desired. The Membership Advisory Committee is interested in promoting membership in all auxiliaries, the Aids to Auxiliaries Committee offers help on programs and projects, and the Public Relations Media Committee has information on the different ways auxiliaries may assist the American Veterinary Medical Association's public relations program.

The national auxiliary celebrated its fortieth birthday this year. So, unlike the political structure with small local units originating first, this federation began at the top. Now effort is being made to promote auxiliaries at the grass root levels where the members live and work. Future officers need to have work experience in not only state and provincial but regional and student auxiliaries as well. So the interest is very genuine in doing everything possible to promote membership in all auxiliaries.

The veterinarian doesn't belong to a union as does the laborer but, instead, he belongs to professional veterinary associations on the local, state, provincial, and national plane. The auxiliaries to these groups are to provide assistance to the associations in whatever form needed, besides serving the worthwhile purposes of furnishing programs and fostering friendship among its own members.

Together, all the auxiliaries form a TEAM, with each organization needing more individual members who are active participants. Teamwork brings results in organizations as well as in basketball. Our national team is made up of individual members who, in turn, belong to affiliates, whose representatives, in addition to the elected officers, form the governing body of the organization. In the House of Representatives of the American Veterinary Medical Association, just constituent associations have delegates and the voting power of each depends on the numerical membership in each constituent group, so the delegate from a small organization has just one vote; from a larger group, the delegate may have five votes. The Auxiliary differs, in that each affiliate has just one vote; consequently the states with more affiliated regionals have more representation, so more interested women have the responsibility of representing their membership and determining the policies of the federation.

The advancement of the whole veterinary profession is the aim and purpose of the Auxiliary. Individual wives of veterinarians banded together in auxiliaries add strength and unity to the profession.

Any organized, unaffiliated auxiliary interested in becoming a working member of the team is invited to contact President-Elect Mrs. E. A. Woelffer, 115 Woodland Lane, Oconomowoc, Wis., for further information.

s/Mrs. Frank R. Booth, *Recorder.*

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COMING MEETINGS

Arizona Veterinary Medical Association. Annual Meeting. Hiway House Hotel, Phoenix, Ariz., Dec. 1-3, 1957, at 2:00 p.m. on December 1. Keith T. Maddy, corresponding secretary.

American Association of Equine Practitioners. Annual meeting. La Salle Hotel, Chicago, Ill., Dec. 16-17, 1957. M. L. Scott, 42 W. Market St., Akron 8, Ohio, executive secretary.

Pennsylvania, University of. Fifty-eighth annual conference of veterinarians. Jan. 7-8, 1958, School of Veterinary Medicine, University of Pennsylvania, Philadelphia. Dr. James Mark, chairman.

Cornell University. Annual conference for veterinarians. New York State Veterinary College, Ithaca, Jan. 8-10, 1958. W. A. Hagan, dean.

Ohio State Veterinary Medical Association. Seventy-fourth annual meeting. Netherland Hilton Hotel, Cincinnati, Jan. 8-10, 1958. Harry C. Sharp, executive secretary.

Oklahoma Veterinary Medical Association. Annual meeting. Hotel Lawtonian, Lawton, Jan. 9-10, 1958. Mrs. Larma Bennett, 2805 S. W. 51 St., Oklahoma City, executive secretary.

Kansas Veterinary Medical Association. Annual convention. Hotel Broadview, Wichita, Jan. 12-14, 1958. K. Maynard Curts, 5236 Delmar Ave., Kansas City 3, Kan., secretary.

Tennessee Veterinary Medical Association. Annual meeting. Hotel Andrew Jackson, Nashville, Jan. 12-14, 1958. H. W. Hayes, 5009 Clinton Pike, Nashville, secretary-treasurer.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Des Moines, Jan. 14-16, 1958. F. B. Young, Waukee, Iowa, secretary.

Indiana Veterinary Medical Association. Annual meeting. Hotel Severin, Indianapolis, Ind., Jan. 15-17, 1958. L. M. Borst, 3315 Shelby, Indianapolis, secretary.

Intermountain Veterinary Medical Association. Annual meeting. Hotel Utah, Salt Lake City, Jan. 16-18, 1958. R. A. Bagley, 4600 Creek View Dr., Murray, Utah, secretary.

Minnesota Veterinary Medical Association. Annual meeting. St. Paul Hotel, St. Paul, Jan. 20-22, 1958. B. S. Pomeroy, School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn., secretary.

Michigan State University. Thirty-fifth annual postgraduate conference for veterinarians, Jan. 22-23, 1958. College of Veterinary Medicine, Michigan State University, East Lansing. W. W. Armistead, dean.

Louisiana State University. Annual veterinary conference. Louisiana State University, Baton Rouge, Jan. 28-29, 1958. W. T. Oglesby, Baton Rouge 3, La., head.

North Carolina State College. Conference for veterinarians. North Carolina State College, Raleigh, Jan. 28-31, 1958. C. D. Grinnells, chairman.

Oregon Veterinary Medical Association. Winter meeting. Portland, Jan. 31-Feb. 1, 1958. Edward L. Holden, P. O. Box 445, Oswego, secretary.

Wisconsin Veterinary Medical Association. Annual Meeting. Pfister Hotel, Milwaukee, Wis., Feb. 10-12, 1958. Robert Curtis, 419 W. Cook St., Portage, secretary.

Colorado State University. Nineteenth annual veterinary conference. Glover Veterinary Hospital, College of Veterinary Medicine, Colorado State University, Fort Collins, Colo., Feb. 17-19, 1958, Lloyd C. Moss, head Department of Medicine, Colorado State University, secretary.

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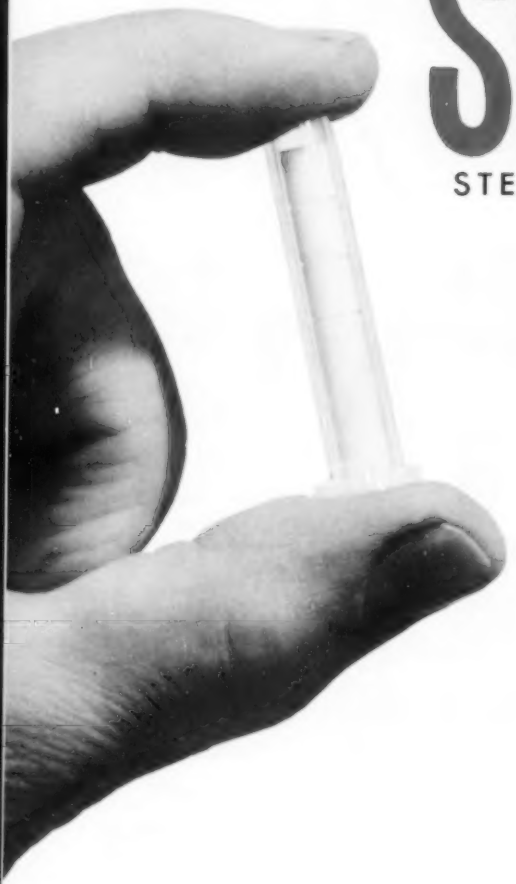

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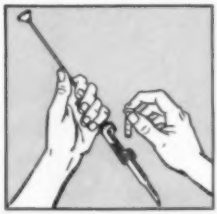
Insert plastic cartridge containing six pellets of Synovex into the chamber of the implanter



Nick through skin of ear with special scalpel. Insert tip of implanter, press plunger to eject all six pellets of Synovex.

Synovex has been cooperatively developed by E. R. Squibb and Sons and Syntex. SYNOVEX is a registered Squibb trademark.

*R. J. Deans, W. J. Van Arsdell, E. P. Reineke and L. J. Brutzler (Michigan Agricultural Experiment Station): The Effect of Progesterone-Estradiol Implants and Stilbestrol Feeding on Feed Lot Performance and Carcass Characteristics of Steers. Journ. Animal Sci. 15:1020, 1956.

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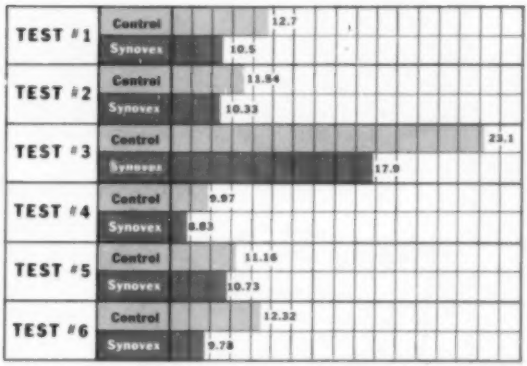
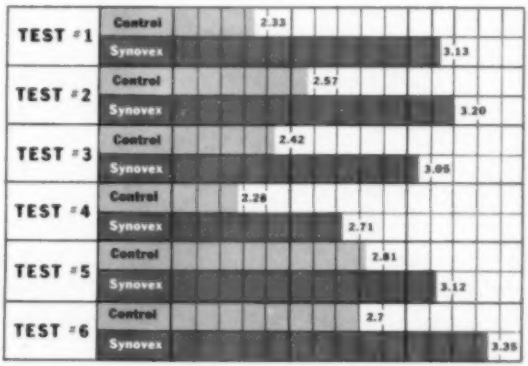
Lbs. of feed per lb. of gain



New Tests Prove Superiority Of Synovex

The two charts at the right prove that Synovex stimulates higher profits as well as higher weight gains. Note dramatic improvement in feed conversion and rate-of-gain when Synovex is used **with a good ration.** These figures prove again that Synovex is today's outstanding product for more profitable feed lot operation.

Gain per head per day



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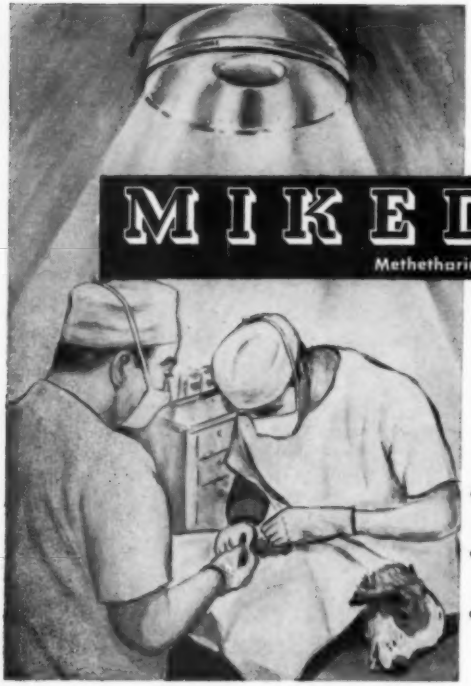

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MIKEDIMIDE has a high therapeutic index, and no signs of toxicity have been observed. Animals can be put to sleep or awakened almost at will, by the alternate use of barbiturate and MIKEDIMIDE without apparent harm to the animal.

MIKEDIMIDE is supplied in 10 ml., 60 ml. and 120 ml. multiple dose vials for parenteral use.

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Illinois State Veterinary Medical Association. Annual convention. LaSalle Hotel, Chicago, Feb. 17-19, 1958. C. B. Hostetler, 1385 Whitcomb Ave., Des Plaines, Ill., executive-secretary.

American Animal Hospital Association. Silver anniversary meeting. Drake Hotel, Chicago, Ill., April 23-26, 1958. Joseph A. S. Miller, president.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December at 3004 16th St., San Francisco, Calif. Mr. Herb Warren, executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third

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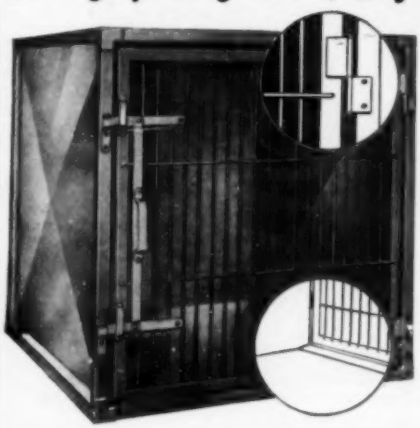

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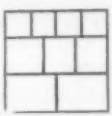

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In cases of otitis in which exudate is present, you will find the drying effect of FURACIN Soluble Powder Veterinary to be most beneficial.

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*Belloff, G. B.: Calif. Vet. 9:16 (Nov.-Dec.) 1956.

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What Is Your Diagnosis?

Make your diagnosis from the picture below—then turn the page ▶

Figure 1

History.—A female Setter, 9 years old, had been used extensively in field trials. For six months before the radiograph was taken, enlargements had been developing on the dorsal surface of the distal extremity of the radius (carpal joint) of both legs, especially of the right one. The enlargements were hard and attached to the underlying tissue. After exercise, the dog showed lameness in the forelegs but especially on the right leg. After each resting period the dog moved soundly.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Osteoperiostitis with a possibility of a calcifying tenosynovitis.

Fig. 2—Radiograph of forelegs of a dog showing osteoperiostitis with a possibility of calcifying tenosynovitis (arrows).

Comments (Dr. James H. Yarborough).*—My diagnosis is osteoperiostitis with possibility of a calcifying tenosynovitis. The lesion may or may not be a part of the bone. I believe this could be determined on physical examination.

This condition occurs frequently in old hunting dogs and occasionally in racing dogs. Since the elimination of stationary wooden hurdles, the incidence in Greyhounds is practically nil.

I have dissected several such involved areas. In most of these, the periosteum was involved. In some, calcium deposits in the tendons or synovial sheath, or both, were observed.

This condition is often seen on the distal end of the large metacarpal bone and the distal end of the radius of race horses. Most of the cases in dogs apparently are due to trauma, as in horses and cattle. The probable reason for this condition in dogs, especially hunting dogs, is that they run through the brush and jump over objects; the affected areas are thus frequently traumatized by external forces.

It has been my experience that if taken in time, rest and injections with hydrocortisone in and around the area will give relief and may restore these animals to usefulness.

This case was presented by members of the staff of the Niles Small Animal Hospital, Niles, Ill.

*Dr. Yarborough is a small animal practitioner in Miami, Fla.

AVMA Research Fellowships Available

The Research Council of the AVMA announces the availability of a number of fellowships for postgraduate training for the academic year, 1958-1959.

The recipient of a fellowship must be a veterinarian and a citizen of the United States or Canada. Veterinary students who expect to graduate at the end of the current school year and who wish to follow a career in research may apply for a fellowship.

The latest date for filing the completed application is Feb. 15, 1958. Approximately one month is required for processing completed applications after receipt by the secretary of the Council. Qualified persons should secure and submit applications as early as possible to insure their file being complete for presentation to the Committee on Fellowships.

The Committee on Fellowships of the Research Council will meet in March to consider applications, and the awards will be announced soon afterward. The stipend will be determined in each case by the needs of the individual, the location of the school in which he proposes to work, and other factors. In general, the stipends range from $100 monthly and upward.

Any qualified person interested in graduate training may obtain application blanks and other information by writing to Dr. C. H. Cunningham, secretary, AVMA Research Council, Department of Microbiology and Public Health, College of Veterinary Medicine, Michigan State University, East Lansing, Mich.

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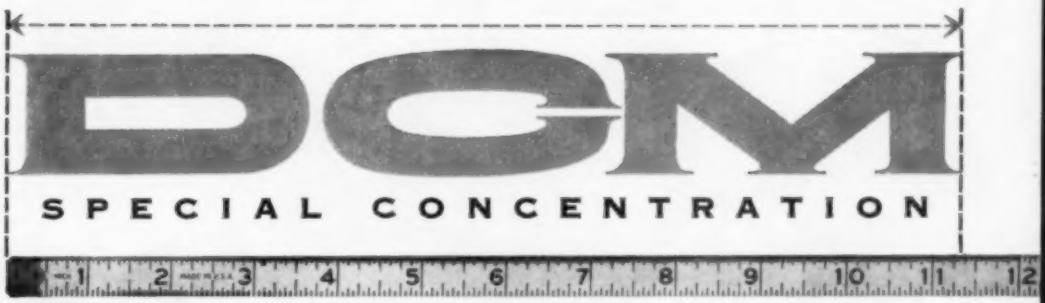

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. . . Same Basic Unit can be Used for Front or Trunk Mounting

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Monday of each month. R. C. Lawson, 40-40 El Camino, Palo Alto, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

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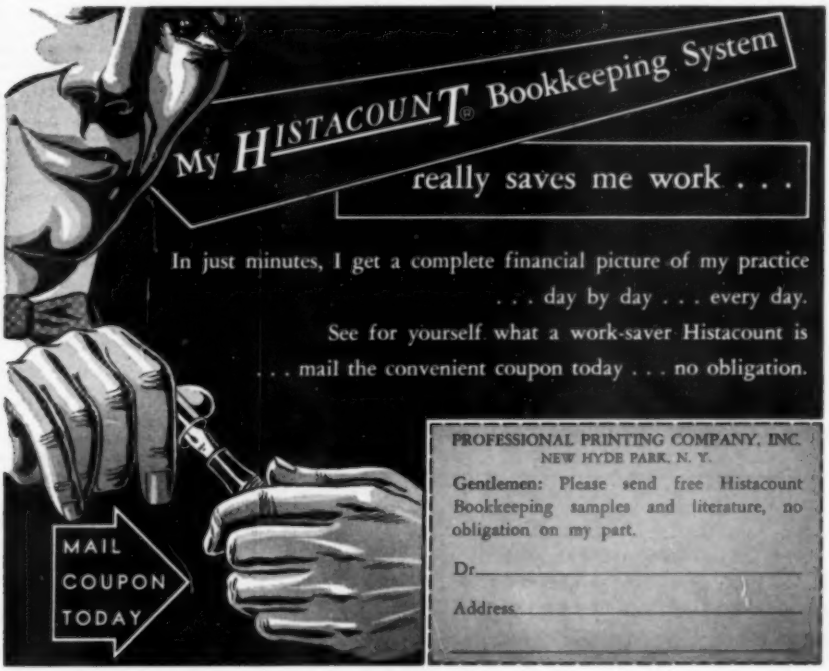
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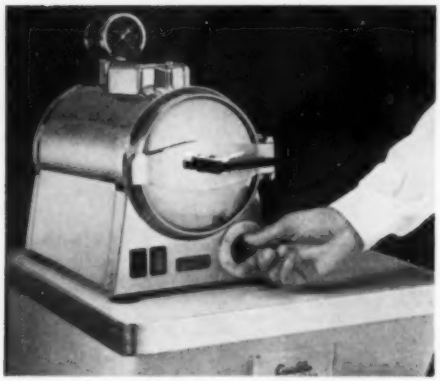

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Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, in St. Petersburg. R. L. Brutus, 336 E. 15th St., Hialeah, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month, at 6:30 p.m., usually in Cedar Rapids, Iowa. William Rugger, Oxford, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa,

6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

KANSAS—Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankford, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Chester R. Pledge, 4249 Peck St., St. Louis 7, Mo., secretary.

Kansas City Veterinrry Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College. Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly, Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Thursday of each month in the Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. James R. Burgess, Rockingham, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

Stark County Veterinary Association, the second Monday of each month. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

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Willamette Veterinarian Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary

PENNSYLVANIA—Del-High Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the Second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P.O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

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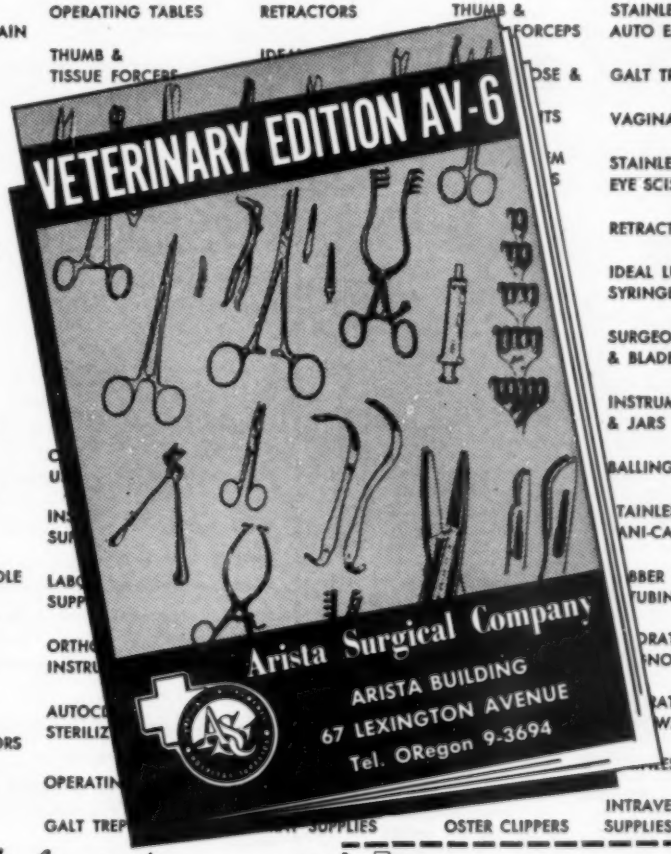

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